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# THE HEART

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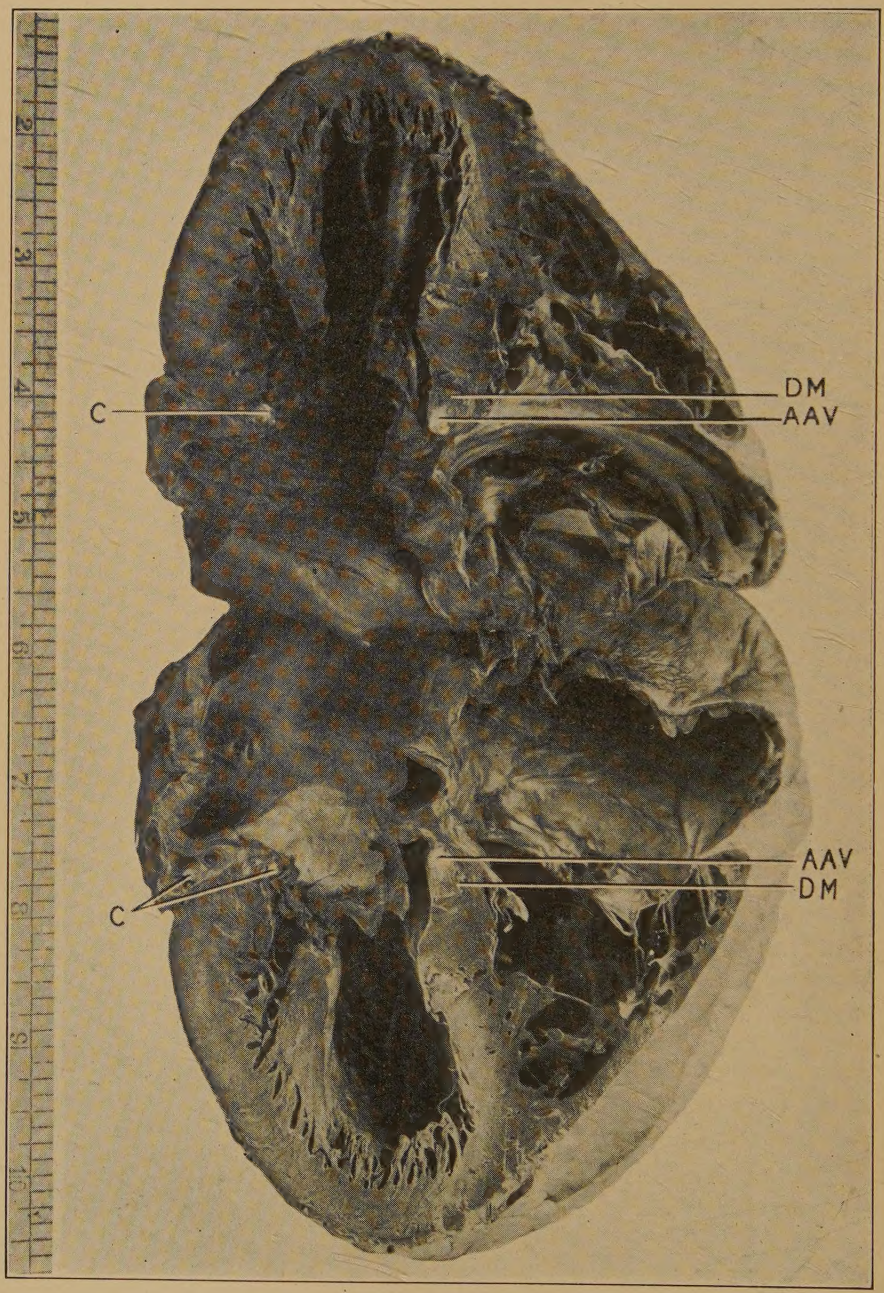


Photograph of heart showing sclerosis of the coronaries (C) and of the artery of the auriculo-ventricular node (A.A.V.). The artery is surrounded by a zone of degenerated musculature (D.M.).

For details with electrocardiogram, see Case 108, Cardiovascular Clinics.

(*Frontispiece*)

# THE HEART

## ITS PHYSIOLOGY, PATHOLOGY AND CLINICAL ASPECTS

BY

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TO MY WIFE

IN TRIBUTE TO HER CONSTANT INSPIRATION AND HELP
THIS BOOK IS AFFECTIONATELY DEDICATED

# PREFACE

The rapid epoch-making advances in cardiology in recent years, with consequent broadening scope of the subject have inevitably resulted in a very voluminous literature. As a result, considerable confusion has arisen as each writer stressed the particular points he wished to bring out, but without sufficient correlation with the larger clinical and practical aspects of cardiology. The importance of some of the valuable contributions to the knowledge of the subject has sometimes been lost sight of because couched in highly technical terms incomprehensible to readers not especially trained in cardiology. The time is therefore ripe for a monograph that shall completely embrace this special field, critically weigh the value of the newer contributions to cardiology, and at the same time present the subject in a manner that can be clearly grasped by the general reader. These have been the main purposes of the author in this book. It was with this in view, that the author has combined the many graphic phases of the subject such as electrocardiography, polygraphy and orthodiascopy with the older and customary methods of clinical examination. Realizing that the necessary instruments are not always at hand for the student or practitioner, and that the necessary technical knowledge for the use of such instruments may not have been acquired, the author has attempted to express in simple language the fundamental principles upon which these instruments are based, together with a sufficiently detailed description of their use. Similarly the meaning of the graphic curves that are obtained is presented in a readily comprehensible manner. The author lays special emphasis throughout on his view that as soon as the basic principles underlying graphic registration and fluoroscopy have been grasped by the reader, proper diagnosis of the cardiac arrhythmias as well as physical diagnosis can usually be arrived at even without the newer instrumental methods. Furthermore the author holds that the clinical value of lessons derived from graphic curves is often less than that to be obtained from the older time-honored non-instrumental methods.

As may be seen by the Table of Contents, many practical questions continually arising in the practitioner's and student's daily routine are considered in this work, and an effort is made to meet many of his perplexing problems in cardiology. Therapeusis, diagnosis with and without instruments, graphic methods, arrhythmias, physiology, cardiac disease (including syphilis), cardiac neurosis, the heart in various extra-cardiac conditions, diet, exercise, etc., are fully dealt with. Embryology, physiology, pathology and the conduction system are given proper emphasis. The newer viewpoints of cardiac function in terms of respiration, blood oxygenation and blood chemistry have also been incorporated.

An original feature of this book is its series of Cardiovascular Clinics in the form of Case Histories. In this manner the every-day phases of the cardiovascular field are covered. These Clinics were given as a course of clinical conferences to senior students of medicine. At first, simple cardiac cases are presented, and the methods and reasons for arriving at the diagnoses are explained. As the conferences proceed more complex cases are discussed, and those questions having a direct bearing upon the cases are given, as well as the author's views as indicated in his responses. Thus the student and practitioner learn how cases should be analyzed and the means by which the diagnosis is made at the bedside. Treatment and the reason for the choice of drugs are given equal consideration. In this manner the practical and clinical side of the cases is presented. Where possible, several similar cases are grouped and discussed, and similarities as well as differences both in physical signs and symptoms are explained.

The aim of this volume has been to present the entire subject of cardiac and cardiovascular disturbances and diseases in a clear, well-balanced manner from the clinician's rather than from a highly specialized viewpoint, and the author hopes that this work will therefore supply a practical, comprehensive reference book for both practitioner and student, as well as for those especially interested in the subject of cardiovascular diseases.

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# PART I

# THE HEART— ITS PHYSIOLOGY, PATHOLOGY AND CLINICAL ASPECTS

## CHAPTER I

### EMBRYOLOGICAL AND ANATOMICAL CONSIDERATIONS—THE CONDUCTION SYSTEM—THE CORONARY SYSTEM—THE SYSTEMIC, PORTAL AND PULMONARY CIRCUITS—CARDIAC NERVE SUPPLY—WEIGHT AND SIZE OF THE HEART—POSITION OF THE VALVES

Embryology as applied to the development of the human heart is important not only because of its intrinsic interest but also because it aids in explaining the vast majority of congenital malformations.

As will be shown later (p. 5), the human heart is essentially a muscular pump made up of intricate systems of fibers and muscular bands, and of valves. The first rudiment of the embryonic heart consists of a simple tube formed by the fusion of two straight tubes on either side of the body; this fusion occurs as the ventral cleft of the embryo closes. The septum of the fused tube disappears so that a simple straight tube constitutes the primary propulsatory apparatus. The tube is lined by a collapsed endothelial lining suspended from the tube wall by fine fibrils. The latter seem constant in the embryo and are regarded by some as the source of the future connective tissue of the endocardium and valves (Mall). The simple muscular cylinder undergoes two simultaneous changes: one, of growth, with elongation, bending and rolling of the cylinder upon itself; the other, of differentiation into cavities. In this process, there is gradually developed an important embryonic construction, the bulbo-ventricular groove. When the embryo reaches 3.5 mm. in length, the heart becomes separated from the mesoderm, the middle layer of the embryo, and is seen as a completed heart tube. By continued growth, twisting and elongation, the cardiac tube becomes S-shaped and is turned on itself in such fashion that the atrial and venous ends are brought close together; what had been the caudal end becomes the left limb of the twisted tube, and the cranial end becomes the right limb (Tandler). At this stage of growth, the heart tube consists of but two

chambers—a single ventricle and a single auricle. The anterior lower part of the twisted cardiac tube consists of the common ventricle (Fig. 1, C. V.) with its aortic bulb (Fig. 1, A. B.); the latter, an important embryological structure, passes upward and to the left, giving off the aortic trunk at right angles.

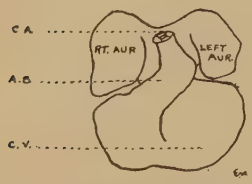



FIG. 1.—Heart of the human embryo 5 mm. long. Front view. × 30. (*Modified from His.*)

The upper part of the common tube consists of a one-chambered auricle (Fig. 1, C. A.), with the sinus venosus behind and to its left. The common auricle is lined throughout with endothelium composed of a solid strand of cells suspended by the delicate fibrils already mentioned. In the later stages of embryonic development the single atrial (auricular), as well as the single ventricular cavity become markedly constricted; septa also begin to form then, thus gradually dividing each common cavity into its respective two chambers. The bulbo-ventricular and interventricular grooves become well marked at this time.

The embryonic aortic bulb (bulbus cordis) leads from the right end of the common ventricle to the aortic arches. In the embryo from 4–6 mm. long, the bulb is a thick walled muscular tube passing to the left and upward. It is lined with endothelium presenting certain endocardial thickenings. These are the so-called proximal and distal bulbar swellings, rudiments of the semilunar cusps and of the lower part of the aorta-pulmonary septum. Thus the human bulbus arteriosus represents developmentally an independent chamber with muscular walls and valves. Later the bulbus disappears, the proximal portion fusing with the ventricular wall. The distal portion becomes elongated and loses its musculature, to become the primitive aortic trunk.

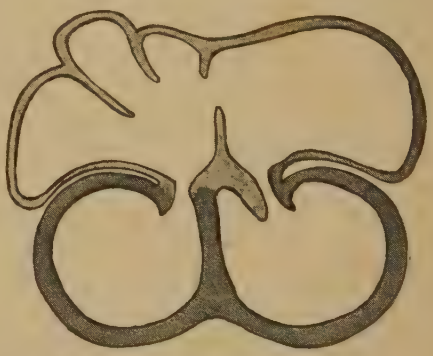


FIG. 2.—Formation of the fetal septa. (*After Aschoff.*)

### DEVELOPMENT OF THE INTERAURICULAR AND INTERVENTRICULAR SEPTA

The interauricular septum is formed partly by the fusion and partly by the disappearance of two separate partitions. The one—the *septum primum*—at about the fourth week begins from the upper and posterior wall of the auricle; it projects downward and forward toward the ventricular cavity but does not completely divide the one-chambered auricle. The hiatus thus formed in the rudimentary interauricular septum is called the *ostium primum*. Later, another opening is formed by the thinning of the *septum primum* itself: this is called the *ostium secundum*. Thus from the primary septum (*septum primum*), which only partially divides

the common auricle, two ostia are formed. A second partial partition—the *septum secundum*—arises much later; this also springs from the upper right auricular wall and is parallel to the *septum primum*. In its downward growth, the *septum primum* partially covers the *ostium secundum*, thus giving the latter a valvular character and forming the *foramen ovale* of fetal life. The persisting portion of the *ostium secundum* found in the adult heart is known as the *annulus ovalis* (Fig. 12). The depression in the septal wall of the left auricle of the adult known as the *valvula foraminis ovalis* (Fig. 12) represents the remains of the fetal *septum primum*.

The interventricular septum begins at about the fourth week; it springs from the inferior wall of the ventricle. With continued growth, the posterior portion of the septum fuses with the corresponding walls of the ventricle, its anterior projection with the anterior ventricular wall, and the medial part with a projection of the aortic septum (q. v.). It is this partition which finally divides the common trunk into the aorta and pulmonary artery. At about the same time, a constriction appears upon the ventricular surface of the heart. This—the interventricular groove—is at first more prominent at the apex and becomes shallower as it courses upward. It marks the external division between the right and left ventricles, and corresponds to the development of the interventricular septum. In this manner, the ventricles and their respective vessels are finally separated into right and left chambers with their corresponding venous and arterial ostia. The junction of the prolongation of the aortic with the interventricular septum forms the *pars membranacia* or the undefended space of the adult heart. It is situated immediately beneath the aortic cusps. As its name indicates, it is membraneous, thin and transparent, and contains no musculature. It is best demonstrated in the adult heart by opening both ventricles of the isolated organ, and then holding the heart suspended by its great vessels: the undefended space then becomes visible as a thin transparent membrane beneath the aortic cusps, and forms the upper part of the interventricular septum in this organ.

## THE AORTIC SEPTUM

The study of the development of the aortic septum is important because congenital malpositions and malformations of aorta and pulmonary artery occur comparatively frequently. The truncus arteriosus—the original common embryonic efferent vessel of the common ventricle—becomes divided into aorta and pulmonary artery by the formation of the aortic septum. Three components are concerned in the formation of this septum—the *septum aorta-pulmonale* (the aorto-pulmonary septum proper), and endocardial elevations in the aortic bulb (*bulbus cordis*) grouped and known as the distal and proximal bulbar swellings. These bulbar swellings later develop into the bulbar septa. The *truncus arteriosus* is thus for a time subdivided as follows: distally by the *septum aorta pulmonale;* mesially by

the distal bulbar septum, and proximally by the proximal bulbar septum. Finally the three septal components—the *septum aorta—pulmonale* and the two bulbar septa coalesce to form the aortic septum. In this manner the *truncus arteriosus* (common arterial trunk) is separated into the two great arterial trunks—the aorta and pulmonary artery. This occurs at about the seventh week of fetal life, at which time the heart is also divided into its various future chambers.

During the period in which the various partitions and septa are being formed in the cardiac cavities and in the common arterial trunk, tissue differentiation is also proceeding by the continued growth of myocardium (especially of the ventricles) and of endocardial thickenings. The myocardium in the spurious or false interauricular septum forms a stray bundle. Muscular ridges—the rudiments of the future *musculi pectinati* (Figs. 11, 12) also form at this period. Musculature likewise forms in the horns and transverse portions of the right and left sinus. The auricular and ventricular musculature is still continuous at this period. In the ventricular musculature, two layers can be distinguished at this stage; a peripheral cortical, and a central trabecular layer. The development of the various muscular layers of the heart will be taken up later.

## DEVELOPMENT OF THE VALVES

The epithelium lining the heart consists of a single layer of epithelial cells. The entire ring of the primary *foramen ovale* is likewise covered by an endothelial layer. The atrial (auricular) endocardial cushions continue their growth downward. At the same time, however, they become gradually undermined by the ingrowing trabecular musculature of the ventricle: they thus become sharp-edged and finally project freely into the ventricular cavity. In the aortic bulb, a similar process proceeds in its bulbar swellings, structures which resemble the endocardial cushions histologically. Here also, at about the level of the atrial canal, there develops a muscular trabecular structure which becomes thicker in texture as it approaches the ventricle. Hence at this stage of their formation, all the rudimentary valves are composed of endocardial projections and cushions, and of musculature. Gradually there is differentiation into valve cusps, *chordae tendinae*, and papillary muscles in the auriculo-ventricular valvular structure, and less marked differentiation in the formation of the semilunar valves.

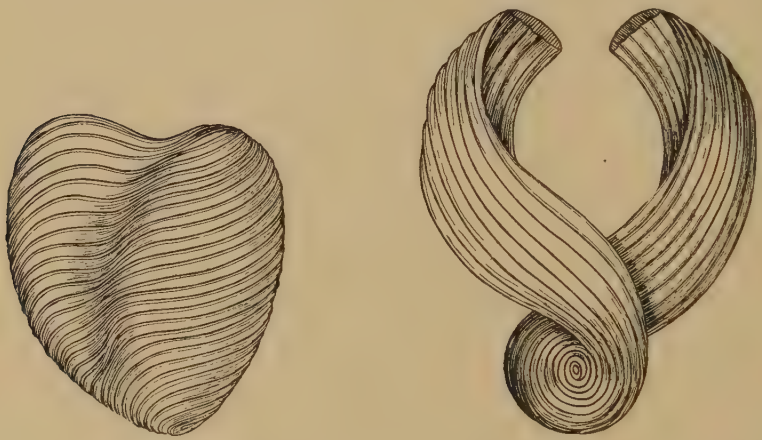
After the division of the heart into its respective chambers, and after the formation of the aortic septum, the right horn of the *sinus venosus* becomes fused with the right auricular wall, and the *valvula venosa sinistra* disappears. The persisting portion of the *valvula venosa dextra* remains as the Eustachian valve (Figs. 11, 12) which, corresponding to its origin, is continuous above and behind with the *crista terminalis* (Figs. 11, 12) and ends below at the coronary sinus. The pulmonary veins are constituted from the original single venous trunk.

**Development of the Cardiac Musculature.**—For the understanding of the complicated muscular architecture of the adult heart, it will be necessary to follow in some detail its embryonic development. Most of the bundles of the heart in the young embryo run circularly; this circular arrangement, however, does not apply to the ventricular apex. The anterior circular group, starting at the bulbo-ventricular groove, passes from the bulb, to run circularly and penetrate the medial side of the left ventricle; this is the sino-spiral bundle. The posterior circular muscle fibers leave the surface of the ventricle and enter the rudimentary inferior septum: this posterior bundle is known as the bulbo-spiral. Even at this stage of development (embryo 10 mm. long) the bundle layers, as they sweep over the apex of the heart, run in such a direction that they form a vortex at the apex. In early embryonic life the circular bundles (the sino- and bulbo-spiral)—are found at the base of the heart. With the downward growth of the ventricles these bundles are pushed downward, so to speak, and the direction of the muscle bundles become more complicated. There are posterior fibers which cross diagonally to the apex of the left ventricle anteriorly, thus forming the anterior face of the apical whorl or vortex. Beneath the sino-spiral bundle are loops encircling the left ventricle, to end posteriorly. They enter the septum from behind and constitute the bulbo-spiral fibers.

Thus, to recapitulate, the simple heart tube is composed chiefly of circular fibers. With the bending of the heart tube upon itself somewhat later, the muscular bundles at the base retain their circular arrangement. The muscle bundles in the apical region undergo changes corresponding in direction to changes in the contour of the cardiac tube. The anterior fibers originating in the bulbo-ventricular groove in front, pass around the anterior surface of the left ventricle to the ventricular septum high up posteriorly, immediately beneath the interventricular foramen. The posterior bundles originating in the bulbo-ventricular groove encircle the right ventricle and pass into the heart in front of the anterior longitudinal sulcus. With the continued growth of the ventricles downward, these two encircling strands are also pushed downward, and finally interlace at the apex of the left ventricle. The fibers from the bulb constitute the bulbo-spiral band and the posterior horn of the apical vortex; those arising from the sinus side form the sino-spiral band and the anterior horn of the apical vortex (Mall).

**Muscular Architecture of the Adult Heart** (Fig. 3).—In the auricular musculature there is a superficial muscular layer which runs transversely and encircles both auricles. Each auricle also possesses a relatively independent system of fibers which runs at right angles to the superficial layers. The course of the ventricular fibers and layers is quite complex and is as yet a matter of uncertainty. According to the most reliable studies, the fibers on the ventral surface arise from tendinous rings and membranes at the base of the heart; here they form a vortex, pass into the interior of the left ventricle to the septum, and connect with the papillary muscles. They

thus turn on themselves toward the base and form spiral loops, which, when contracting, approximate base and apex, and at the same time rotate the apex clockwise from left to right. Mall divides these "superficial fibers" into two groups: the superficial bulbo-spiral and the superficial sino-spiral. The former belong chiefly to the left ventricle. They arise from the conus to the left of the aorta and left ostium venosum, proceed spirally, penetrate the anterior aspect of the left ventricle and end in the septum and posterior aspect of the ventricle, at that point they connect with the posterior papillary mus-



*A* *B*

FIG. 3.—*A* and *B*. Muscular architecture of the human heart. Exact reproduction from Tractatus de Corde, by Richardo Lower, M.D., published in 1649, showing the general arrangement of the layers (*A*) and the arrangement of the special fasciculi *B*.

cles. Some of the deeper fibers of this layer encircle the lower part of the ventricle and pass upward, to end at the base of the heart. The superficial sino-spiral fibers arise mainly from the posterior aspect of the heart in the neighborhood of the right ostium venosum, proceed spirally (though more transversely than the first group) to the apex across the anterior surface of the right ventricle. They penetrate the interior of the left ventricle and terminate on its anterior surface and in the papillary muscles, especially the anterior. Beneath the superficial layers of the bulbo- and sino-spiral systems lie similar deep layers which run more transversely or circularly. The deep bulbo-spiral layer encircles the left ventricle and ends by way of the septum on the dorsal side of the aorta. Some fibers make a circular loop around the conus at the base of the pulmonary artery. The entire layer makes a strong circular system whose contraction tends to diminish the lumen of the left ventricle. The deep sino-spiral layer originates from the posterior aspect of the left *ostium venosum*, passes transversely to enter the interior of the right ventricle, and then turns upward toward the base. Here some strands pass circularly around the base of the heart and left ostium.

## THE CONDUCTION SYSTEM

Because most of the advance in modern cardiology is based upon our comparatively recent exact knowledge of what is known as the junctional tissue, the bundle of His or the conduction system—a subject to be often alluded to—it is important to study in detail its embryonic beginnings as well as its structure and distribution in the adult heart. Former embryologists regarded the atrio-ventricular conduction system as a new formation. According to Mall, however, it is to be regarded as the remnant of the wall of the atrial canal after its anterior and lateral sides have been broken in the formation of the tricuspid and mitral valves. In various sections of the human embryo, the bundle can be followed as a definite structure commensurate with the breaking down of the atrial wall. The conduction system is plainly visible in an embryo 20 mm. long (Fig. 4). Not only does the growth of the endocardial cushions previously described play a part in the final breaking down of the atria but there is also decided ingrowth of the connective tissue of the epicardium in the neighborhood of the atrio-ventricular groove; connective tissue prolongations extend into the anterior and posterior longitudinal sulci and into the auriculo-ventricular valves. Posteriorly this circular connective tissue surrounds the atrio-ventricular bundle so that the latter becomes wedged between the posterior

FIG. 4.—The atrio-ventricular conduction system in the fetus. (*After Mall.*)

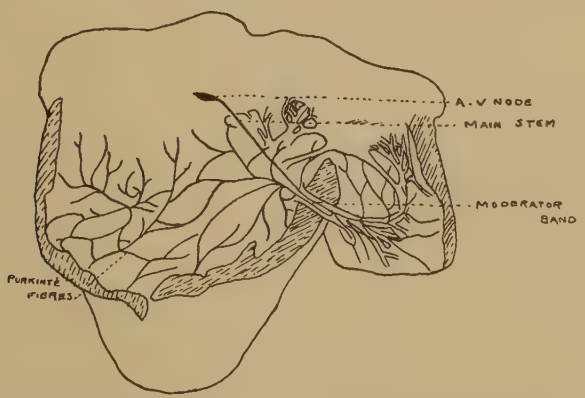



FIG. 5.—Right ventricle of ox heart, showing the auriculo-ventricular node (A-V node) and the main stem. The right branch is seen running along the "moderator band." (*Modified from Tawara, "Das Reitzleitungssystem des Säugethierherzens."*)

endocardial cushion and the outer connective tissue. This, the survival of the embryonic muscular connection between the auricles and ventricles, represents the future atrio-ventricular conduction system (the bundle of

His)—later to become differentiated and innervated. Its embryonic position corresponds to its position in the adult.

In order to have a definite conception of many of the physiological functions of the heart, as well as of its patho-physiology, it is important to have a clear picture and even a visual image of what constitutes the conduction system in the adult heart. The conduction system has been variously named the atrio-ventricular conduction system, the auriculo-ventricular conduction system and the junctional tissues. But no matter how called, the outstanding fact to be remembered is that the conduction system forms a continuous strand of specialized fibers encased within special sheaths and

FIG. 6.—Left ventricle of ox heart, showing conduction system. (*Modified from Tawara.*)

having special attributes. The entire system may perhaps be likened to a slender uprooted and inverted Y-shaped tree stripped of its leaves (slender, for the width of the main stem in the adult heart is approximately 1 mm.). The tree root-clod would then represent the node of Tawara, the origin of the conduction system: the tree trunk before its division, the main bundle of the conduction system (the bundle of His); the two limbs of the Y, the main branches that go to the right and left ventricles, respectively; and the smaller branches and twigs of the tree, the twigging branches and final aborizations of the conduction system.

In the adult, for purposes of anatomical identification, the various parts of the conduction system (Figs. 5, 6, 7, 8) are differently named, but as already stated, they together form one continuous strand. The node of Tawara —the beginning of the junctional tissue—and the bundle of His constitute the major part of the conduction system before its division. The node and bundle lie immediately beneath the endocardium in the lower part of the right auricle, slightly above the level of the ventricle, and about midway between the opening of the coronary sinus and the fibrous tissue beneath the aortic cusps (the aortic vestibule or "undefended space" (Figs. 11, 12). The further continuation of the conduction system is called the main stem; this soon makes a hairpin-like division into its two main branches—the right and left (Figs. 5, 6, 7, 8). These branches course respectively on either side of the interventricular septum in a direction roughly parallel to the axis of the

respective ventricular cavities. The right branch is thin and spreads out in a somewhat fan-shaped fashion; the left is more compact, thicker and club-like. In their upper portions both branches are superficial and subendocardial, becoming deeper as they course downwards. At the same time they split into secondary and minor twigs and twiglets as they spread toward the apex (Figs. 5, 6, 7, 8). In the human adult heart, the main stem and main

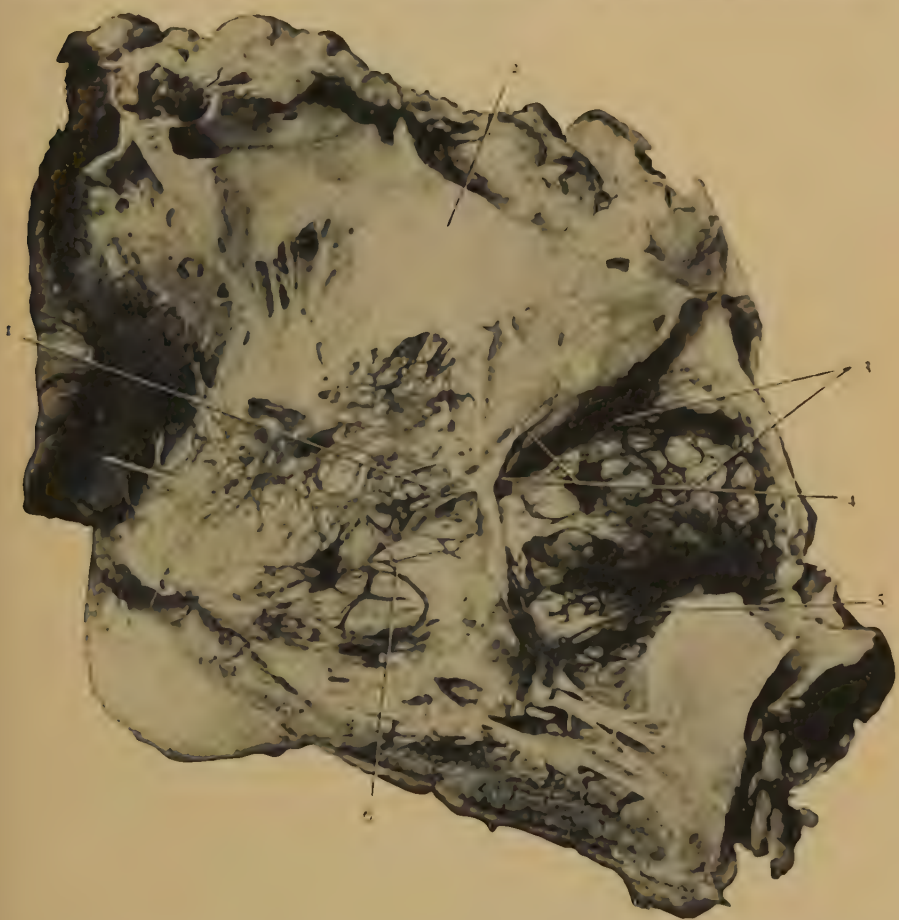



FIG. 7.—1–Branch to base of right ventricle; 2–Tricuspid valve; 3–Conus arteriosus; 4–Right division of A-V Bundle; 5–Branch to conus arteriosus; 6–Branch to apex. (*Courtesy of Nelson's Loose-Leaf Living Medicine.*—After A. E. Cohn, injection specimen of conduction system on the ox-heart.)

branches of the conduction system are sometimes dimly discernable through the shimmering endocardium as separate, paler, non-glistenings tructures. In the ox heart the main branches can be studied macroscopically by first hardening the heart in formalin and then carefully teasing off the overlying endocardium with forceps. If this be done, the left branch will be found to have three main divisions—one to the apex, and one each to the anterior and

posterior papillary muscles. The right branch of the ox heart has one division that goes to the large papillary muscle and venous base, the other forms the "moderator band" (Fig. 5) which supplies the septal papillary muscle and the arterial base (conus arteriosus); from both of these, a division to the

FIG. 8.—1–Aortic valve; 2–Mitral valve; 3–Left division of A-V bundle; 4–Branch to posterior papillary muscle; 5–Branch to apex; 6–Branch to anterior papillary muscle. (*After A. E. Cohen.*) (*Courtesy of Nelson's Loose-Leaf Living Medicine.*)

apex is formed from separate heads. In both human and mammalian hearts the branches finally divide into terminal aborizations (known as Purkinjé fibers in the mammal), which ramify throughout the papillary muscles and probably throughout the entire ventricular musculature. Some of these terminal aborizations run free across the apical portion of the ventricular

cavities; they were formerly regarded as aberrant tendinous strands. All are not purely tendinous, however. Macroscopically, they may be distinguished from tendinous connective tissue structures by their paler and finer texture and non-glistening appearance; and microscopically, final aborizations are found to consist of fine, pale, delicately staining cells.

The main branch and the various subsidiary branches and fasiculi of the conduction system are enveloped in special sheaths of connective tissue. The smaller enveloping sheaths are fairly constant in structure. The main sheath varies somewhat, the connective tissue being at times condensed; at others, looser and almost areolar in texture. The main sheath contains the blood and lymph vessels, and nerves.

**Dissection and Demonstration of the Conduction System.**—In order to dissect out the conduction system, the endocardium over the site of the auriculo-ventricular (A-V) conduction system is gently teased off with forceps after the heart has been washed in water and hardened in a formaldehyde solution. The conduction system is most readily dissected in the ox or calf heart, less readily in the human. After hardening, the heart is first incised with a scalpel near the margin of the interventricular septum. The ventricles are then cut parallel to the latter. In this manner they can be turned back flapwise, and the septal walls of the auricle and the ventricular septum exposed without injury to the conduction system. In oxen the main branch on the right side exists as a separate strand—the moderator band. After exposure by teasing in the manner above described, the structure of the atrio-ventricular (commonly abbreviated A-V) conduction system will be found paler, softer, and more delicate than that of the surrounding musculature. Another method of gross demonstration of the conduction system is by subendocardial injection of the main branches with a fine hypodermic needle and syringe containing a 50 per cent. solution of India ink (Figs. 7, 8). If carefully performed, not only the main branches but the subsidiary divisions as well stand out prominently as darker strands. In this manner, also, corroborative evidence is derived that ventricular contraction begins at the papillary muscles, for the ink may be seen to reach the latter first, and then the base and apex of the heart. For purposes of injection, a No. 1 Luer hypodermic syringe with the finest steel needle is used. By careful multiple small injections of various fasciculi, a complete picture of the main branches with their ramifications and distribution is possible. It can thus be seen that in the left ventricle of a beef heart there are two main strands coursing along the interventricular septum; these form two subsidiary branches which cross the left ventricular cavity and form a network at the base of the papillary muscles; the interlacing fibers however reach only halfway to the apices of these muscles. The network then spreads upward to an area of the ventricles situated beneath the mitral cusps and near the fibrous ring of the mitral orifice. The posterior ventricular wall is also richly supplied by similar superficial interlacing fibers. The septal wall itself is not as abundantly

covered with the superficial network as are the ventricular regions just described. The main left branch at its point of emergence beneath the aortic cusp is flat and superficial. As it forks, the branches become more oval or cylindrical; these course in the trabecular muscular bridges of the ventricular musculature and link together the septal and posterior ventricular walls. Most of the so-called false or aberrant tendinous fasciculi found in the ventricular cavity are actually the final threadlike ramifications of the conduction system. The tendinous-like threads may be divided into three classes: (1) Those containing both the ordinary cardiac muscular fibers as well as the special fibers of the conduction system; these form a majority of the strands. (2) Those made up of connective tissue alone. (3) Those made up of fibers of the conduction system alone (DeWitt).

The main right branch of the atrio-ventricular conduction system of the beef heart also divides and subdivides freely, so that the right ventricle is likewise plentifully supplied with subsidiary branches, networks and anastomoses. The right branch, more deeply embedded subendocardially than the left, is usually cylindrical in form. It courses along the right of the septum and reaches the moderator band, along which it runs until it reaches the anterior papillary muscle. To judge from specimens studied by the injection method, apparently the right branch does not subdivide until it reaches the base of the anterior papillary muscle.

The injected atrio-ventricular node resembles somewhat in form the semilunar ganglion. Besides its continuation into the main stem, and right and left branches already described, there are in the beef heart apparently two other prolongations of the node; one passing upward going in the direction of, and seemingly connected with the sino-auricular node (the node of Kieth-Flack), and the other passing in the direction of the coronary sinus.

The main arterial supply of the auriculo-ventricular conduction system (for convenience called the A-V system) is derived from a special branch of the right coronary. The junctional tissue is supplied and intertwined with medullated nerve fibrils and ganglia. There are numerous ganglion cells—(mono-, bi-, and multipolar)—whose processes pass to adjacent ganglion cells, to nerve fibers in the bundle or through the A-V system, and end in ganglia cells of the bundle or in the muscle plexus. There is an intricate plexus of varicose fibrils around, and in close relation to, the muscle fibers of the bundle. Thus the A-V system, like the sino-auricular node, is a neuromuscular structure. Although the exact distribution of the extrinsic cardiac nerves to the nodes is not known, it appears probable that, regarding the vagus distribution, the sino-auricular (or S-A) node is supplied chiefly by branches of the right vagus, and the A-V by those of the left. There appears to be a similar distribution to the S-A and A-V nodes by the right and left accelerators, respectively.

**The Rhythm Center or Pacemaker** (Fig. 9).—It has required much patient experimentation, and the combined work of physiologist, histologist,

pathologist and electro-physiologist to finally determine the exact site and minute structure of the rhythm center (also called the pacemaker) of the mammalian heart. It may now be definitely set down that the rhythmic periodic impulses which normally excite the mammalian heart have their origin in the sino-auricular node (also called from their discoverers, the node

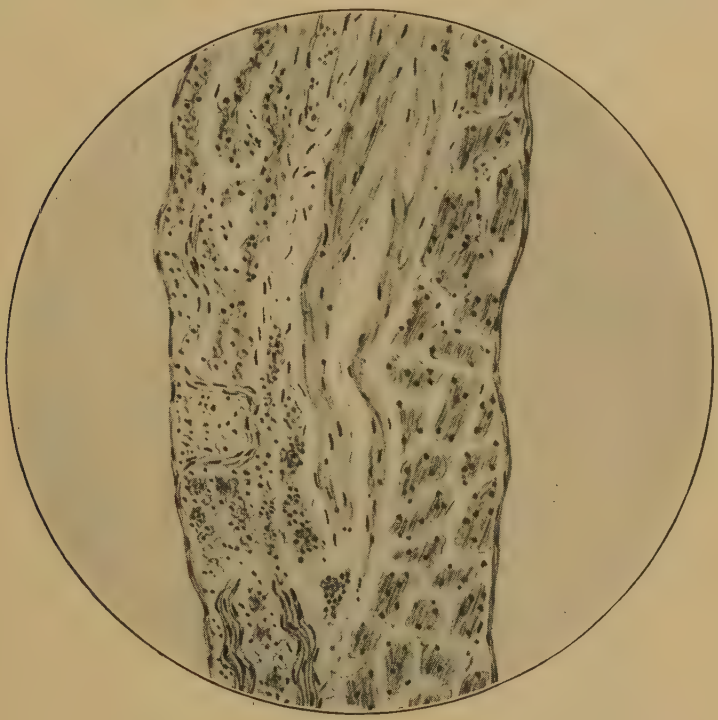


FIG. 9.—Sino-auricular node of pig's auricle. (*After Lewis.*)

of Keith-Flack), and that this node constitutes the normal rhythm center or pace-maker. This bit of specialized cardiac tissue is histologically a neuromuscular structure. It is irregularly pear-shaped with a larger upper, and a somewhat tapering lower end; it is situated in the sinus region, in the groove at the junction of the superior vena cava and right auricle (the *sulcus terminalis* (Fig. 12)) immediately beneath the epicardium. The node is surrounded by branches of the vagus and sympathetic: it contains a plexus of moniliform nerve fibrils and a few ganglionic cells; it is, therefore, histologically considered, a neuro-muscular structure. Its blood supply is derived from a special artery. The structure, arrangement and composition of the node differ materially from the remainder of the cardiac musculature (Fig. 9): the cells are smaller, stain more delicately and are paler; the cross striations are indistinct or may be absent; the nuclei are pale: there is a relative richness of perinuclear sarcoplasm. The cells contain more glycogen

than those of the non-specialized muscle. They do not follow any orderly, layer-like arrangement, but are placed irregularly in a rich stroma of fine connective tissue. A small specialized muscle band connecting the sino-

FIG. 10.—Roentgenogram of the blood supply of the average heart. (*After Gross.*) The coronary arteries and their branches.

auricular node with the remainder of the conduction system has been described (Thorel), but its presence has not been corroborated by other observers.

In early fetal life, the heart lies immediately under the head and is of relatively large size. Later, it becomes a thoracic organ, lying at first vertically, then gradually assuming a more oblique position. The auricular portion with its intercommunication (the foramen ovale) is at first larger

than the ventricle. By means of the ductus arteriosus (ductus Botalli), the blood from the right ventricle and pulmonary artery passes mainly to the aorta instead of to the lungs. To carry on this circulation, the wall of the right ventricle is correspondingly muscular and as thick as that of the left. Toward the end of fetal life, the left ventricle becomes thicker and heavier than the right, and remains so throughout life.

**Coronary Circulation.**—The intra-cardiac blood supply demands detailed consideration, for disease of this arterial system is very common and accounts for many of the symptoms and cardiac changes found in certain types of heart disease. The heart itself derives its arterial supply from the right and left coronaries (Fig. 10). Each arises from the corresponding aortic sinus. The right coronary courses at first between the right auricle and conus arteriosus; it finally runs along the posterior longitudinal sulcus, and, as the posterior descending ramus, almost reaches the apex of the heart. Branches of the right coronary supply the right ventricle, the right atrium and to a lesser extent the left ventricle.

The left coronary is usually the larger of the two. It almost immediately divides into a right descending and a circumflex branch. The former soon runs along the anterior longitudinal sinus toward the apex of the heart; it supplies the interventricular septum, the left ventricle and to a slight extent, the right ventricle. The circumflex branch supplies the left ventricle and auricle.

Contrary to older opinions, anatomical investigations by Spalteholz and more recently by Gross have demonstrated that the coronaries are not end arteries and that they anastomose freely by means of minute branches; the latter are usually found in the cardiac muscle itself, more rarely upon the surface of the heart. The fact that the coronaries anastomose is extremely important, for it explains upon anatomical grounds how some patients with occlusion of even comparatively large coronary branches may continue to live by the establishment of collateral circulation. Gross's research has also established some interesting and suggestive facts regarding the size and distribution of the coronary systems in the various decades of life. Thus in early life, there is not much difference between the right and left coronary distributions, as shown by injected specimens. With advancing years the left gradually gains the ascendancy in the number and distribution of its branches. Finally in the old, there is gradual restriction of the distribution of both coronaries, but particularly that of the right coronary artery. Such anatomical studies may perhaps account for the more serious clinical symptoms occurring in occlusions and disease of that coronary system which is already undergoing retrogression.

The veins of the heart accompany the coronary arteries and their branches, and empty into the right auricle.

The lymph vessels of the heart are very abundant; they are formed from radicals derived from the lymph spaces in the clefts between the muscle

fibers; they accompany the blood vessels in their course and terminate in the thoracic and right lymphatic ducts.

Regarding the systemic circulation mechanically and as a whole (Figs. 11, 12, 13) the primary source of power is of course the heart. But the heart may after all be regarded as a means to an end. The central pumping station

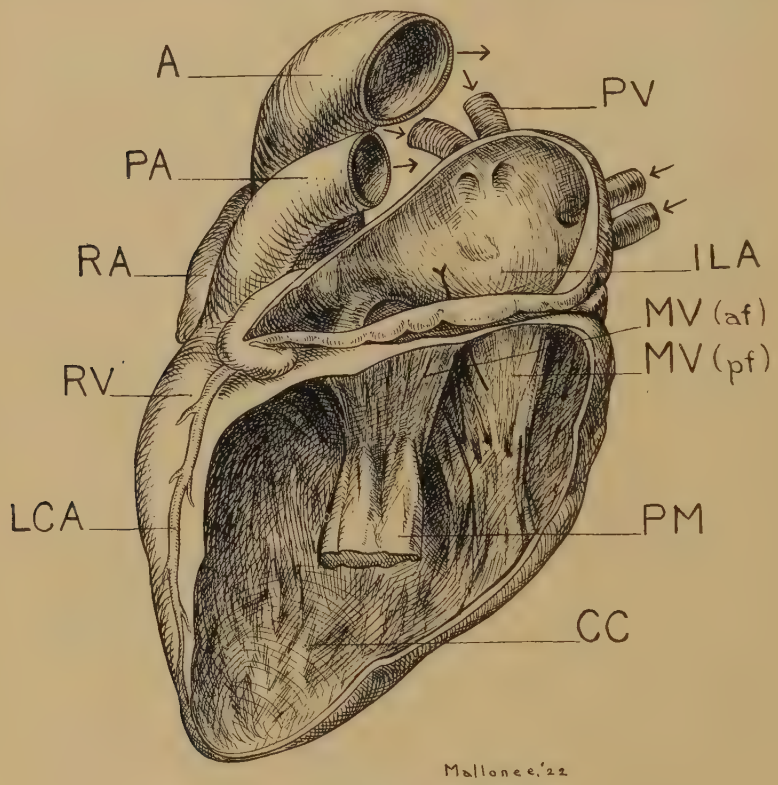



FIG. 11.—The adult heart, left side. (*After Gray.*)
A, Aorta; P.A., pulmonary artery; R.A., right auricle; R.V., right ventricle; L.C.A., left coronary artery; P.V., pulmonary veins; I.L.A., interior of left auricle; M.V. (a.f.), anterior flap of mitral valve; M.V. (p.f.), posterior flap of mitral valve; P.M., detached papillary muscle; C.C., columnæ carneæ.

is designed to carry the blood especially to and through the capillaries, by means of which the growth, nutrition and chemical change of life go on; for the capillaries come into immediate and direct contact with the tissue cells. It is interesting to recall the significant structural adaptation of various parts of the cardio-vascular system to their special purposes. Thus, the larger arteries consist chiefly of elastic tissue; they are readily distensible, can accommodate large quantities of blood, and by their elastic recoil, they propel the blood after cardiac systole has ceased. The arterioles contain many smooth muscle fibers under the control of the vaso-motor system; this com-

bination acts to control the amount of blood flow to the capillary bed. The capillaries consist of cells and basement membrane, so that there is ample opportunity for the exchange between blood and tissue. The veins are thin-walled and distensible, so that, if necessary, they can accommodate large quantities of blood; this is especially true of the splanchnic area. Some of the larger veins have valves in order to assist the return circulation to the right heart.

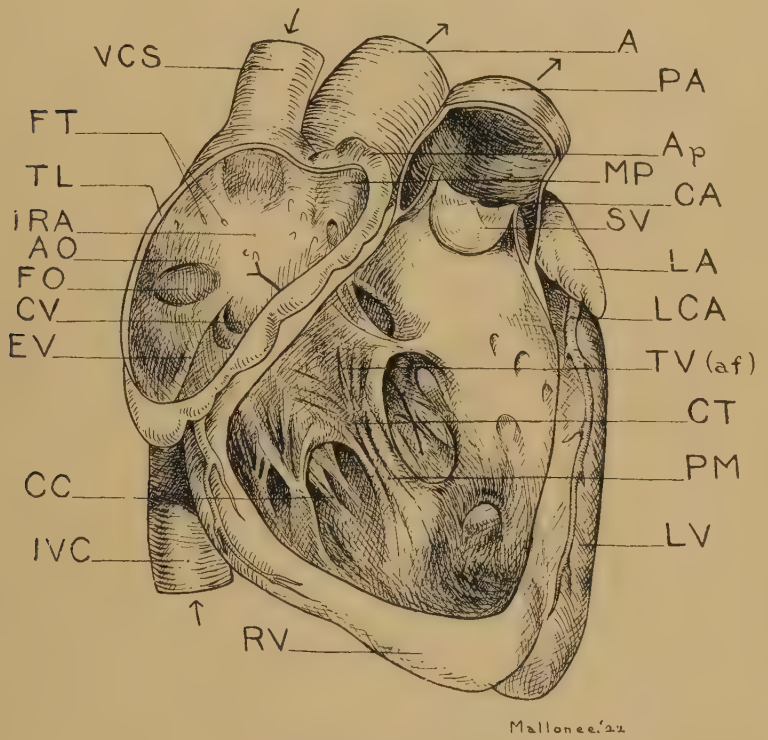
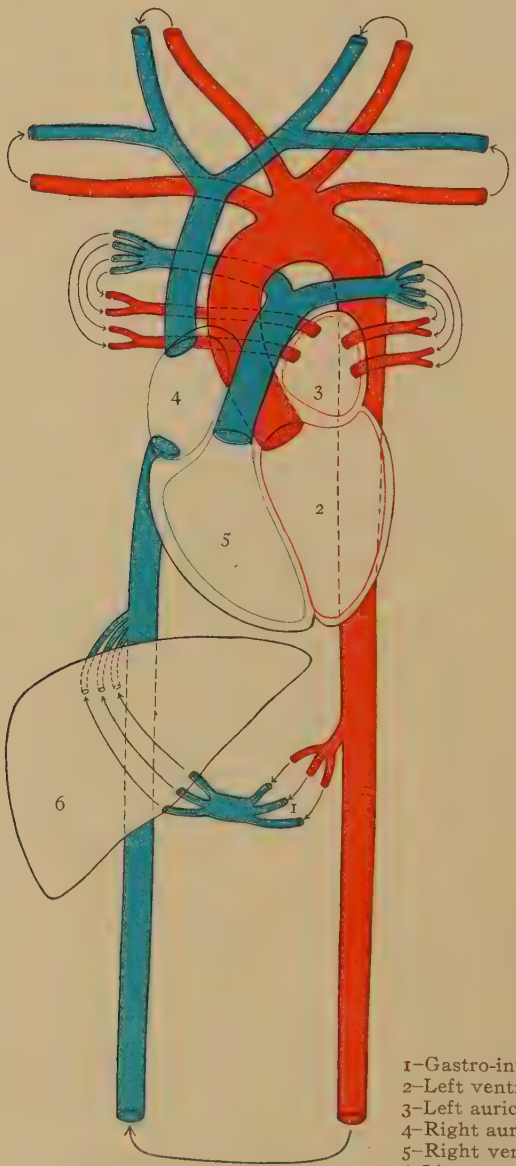



FIG. 12.—The adult heart, right side. (*After Gray.*)

V.C.S., vena cava superior; F.T., foramena Thebesii; T.L., tuberculum Loweri; I.R.A., interior right auricle; A.O., annulus ovalis; F.O., fossa ovalis; E.V., Eustachian valve; C.V., coronary vein; C.C., columnæ carneæ; I.V.C., inferior vena cava; R.V., right ventricle; A, aorta; S.V., semilunar valves; L.C.A., left coronary artery; T.V. (a.f.), anterior flap of the tricuspid valve; C.T., chordæ tendinæ; P.M., papillary muscle; L.V., left ventricle; P.A., pulmonary artery; A.P., appendix; C.A., corpus Arantii.

Two subsidiary systems—the portal and pulmonary—(Fig. 13)—remain to be briefly considered. The portal vein collects the blood from the venous radicals of the intestinal walls and villi. This blood, with its elaborated food products as the result of intestinal digestion, is re-distributed through the liver by means of a second set of hepatic capillaries, to be again collected into the inferior vena cava. In the pulmonary circuit, the venous blood is distributed to the alveoli in the lungs, so that the venous blood loses its surplus




1–Gastro-intestinal canal;
2–Left ventricle;
3–Left auricle;
4–Right auricle;
5–Right ventricle;
6–Liver.

FIG. 13.—Schematic view of systemic, pulmonary and portal circulations.

of carbon dioxide and receives oxygen in exchange. The freshly oxygenated blood is finally returned to the left auricle by means of the pulmonary veins. Thus the pulmonary circuit is completed.

**The Cardiac Nerve Supply** (Figs. 14, 15).—It is necessary to describe in some detail the nerve supply of the heart because of its importance both from the physiological and clinical standpoints. The extrinsic nerve supply is an extremely complicated one. It is rendered more so by the presence of numerous plexuses which themselves intercommunicate and decussate. The extrinsic cardiac nerves are those derived from the vagus and from the sympathetic system. The vagus arises by several roots in the medulla in the furrow behind the olivary body and below the origin of the glossopharyngeus (Figs. 14, 15). It contains not only motor and sensory, but also vegetative fibers. It sends afferent branches to the heart, pharynx, esophagus and stomach, and possibly also to other viscera. The chief physiological function of the vagus on the heart is inhibition. The depressor nerve, the efferent nerve of the heart, has branches which are grouped chiefly about the root of the aorta. It runs as part of the vagus and forms the viscero-sensory tract of the latter nerve. Experimental stimulation of the central cut end of the depressor causes a fall of blood pressure by reflex action upon the medulla. The chief sympathetic afferent nerve of the heart is the accelerator. In mammals, the two most constant branches of the sympathetic system are those which surround the subclavian arteries to form the plexus known as the annulus of Vieussens (*ansa subclavia*). Situated at the base of the heart are the superficial and deep cardiac plexuses. The superficial plexus lies in the concavity of the aortic arch; the deep plexus, between the trachea and aorta. The latter is composed of nerves derived from the sympathetic cervical ganglia, and from the cardiac branches of the recurrent laryngeal and vagus. The branches from the right side of the plexus go to form parts of the anterior and posterior coronary plexuses; they also send a few filaments to the right auricle. The branches from the left side are distributed to the left auricle and compose a large part of the posterior coronary plexus. The superficial cardiac plexus forms the chief part of the anterior coronary plexus.

The posterior and anterior coronary plexuses surround and accompany the branches of the right and left coronary arteries, respectively, and distribute filaments to the ventricular musculature. The coronary plexuses as well as their muscular filaments are richly supplied with ganglia.

Intracardiac ganglia have been found in the auricular wall, at the mouths of the superior and inferior vena cava, and at the origin of the coronary sinus. They have also been found at the level of the auriculo-ventricular junction, especially about the aorta and the pulmonary artery. Scattered ganglia, either as single cells or in small groups, have likewise been observed practically throughout the entire heart. As has already been pointed out, impulses normally originate in the pacemaker (the sino-auricular node), and not, as previously thought, in other ganglionic groups.

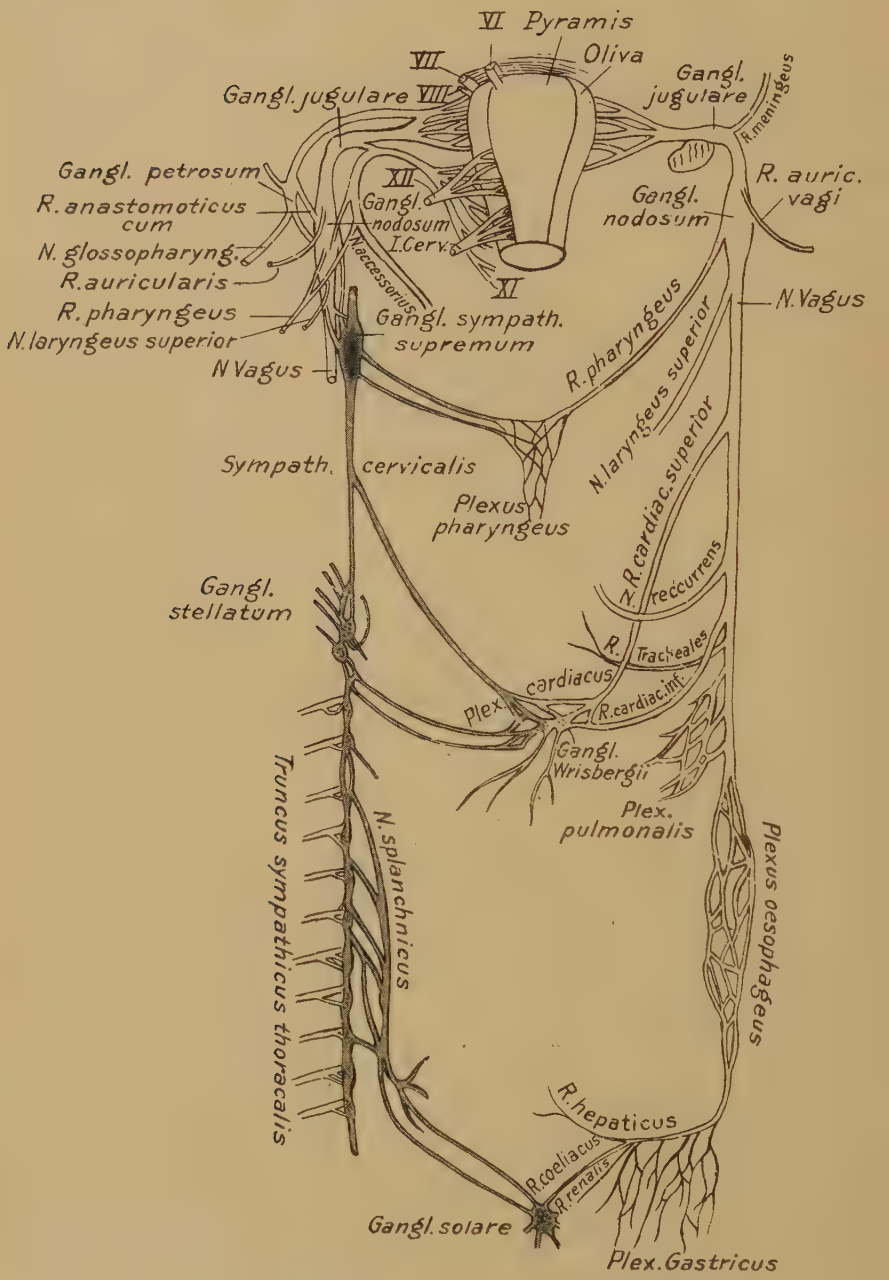



FIG. 14.—Extrinsic nerves of the heart. (*After Muller.*)

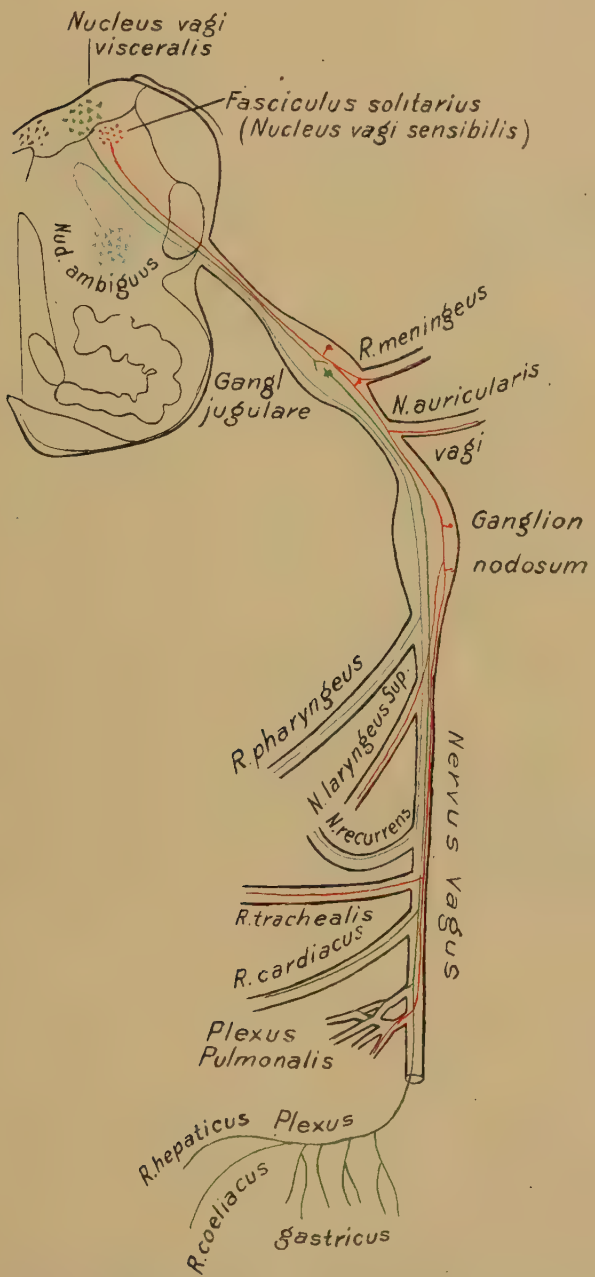



FIG. 15.—Schematic view of the vagus and its branches, and their connection with the sympathetic. (*After Muller.*)

Of special clinical importance and interest is the connection between the extrinsic nerves with the sino-auricular node and the atrio-ventricular conduction system. These two specialized structures are supplied by, and intertwined with, nerve fibrils and ganglia. Regarding the atrio-ventricular conduction system, as already stated, they contain numerous ganglion cells, with one, two or several polar processes; these processes pass either to adjacent ganglion cells, to nerve fibers in the bundle, or directly to the conduction system. Some nerve fibers end in ganglia cells of the bundle or in the muscle plexus. There is also an intricate plexus of varicose fibrils around, and in close relation to, the muscle fibers of the bundle, making the auriculo-ventricular conduction system, like the sino-auricular node, a neuro-muscular structure. Although the exact distribution of the extrinsic cardiac nerves to the nodes is not known, it appears probable that, regarding the vagus distribution, the sino-auricular (S-A) node is supplied chiefly by branches of the right, and the atrio-ventricular (A-V) node by those of the left vagus. There appears to be a similar distribution to the S-A and A-V nodes by the right and left accelerators, respectively.

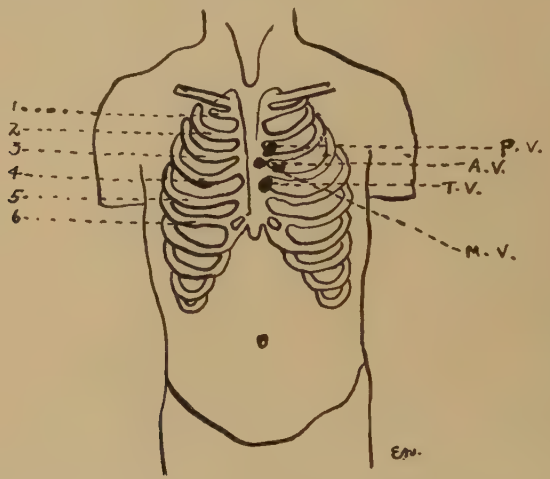



Fig. 16.—Diagrammatic position of the cardiac valves.
P.V., Pulmonary valves; A.V., aortic valves; T.V., tricuspid valves; M.V., mitral valves

**Weight and Size of the Heart—Position of the Valves.**—The average normal adult heart weighs, in the male, from 280 to 360 grams (9½ to 12 oz.); in the female, from 240 to 330 grams (9 to 11 oz.); its proportion to body weight ranges approximately from 1:160 to 1:170. Although hearts vary considerably in size within normal limits, the average length of the adult heart on its longest axis is from 12 to 15 c.m. (4¾ to 6 inches); its greatest breadth, 9 to 11 c.m. (3½ to 4½ inches); thickness, 5 to 8 c.m. (2 to 3 inches). The normal circumference of the various valvular orifices is as follows: the tricuspid, 11 to 13 c.m. (5 to 5½ inches); the mitral 9 to 11 c.m.

(4¼ to 5 inches): the pulmonary, 8 to 9 c.m. (about 3¼ inches); the aortic, 7 to 8 c.m. (about 3¼ inches). The adult heart lies behind the lower two thirds of the sternum. The methods for determining the position of the valves with the heart *in situ* by frozen sections, and also by coating the valvular surface with lead and subsequently taking radiographs, have not

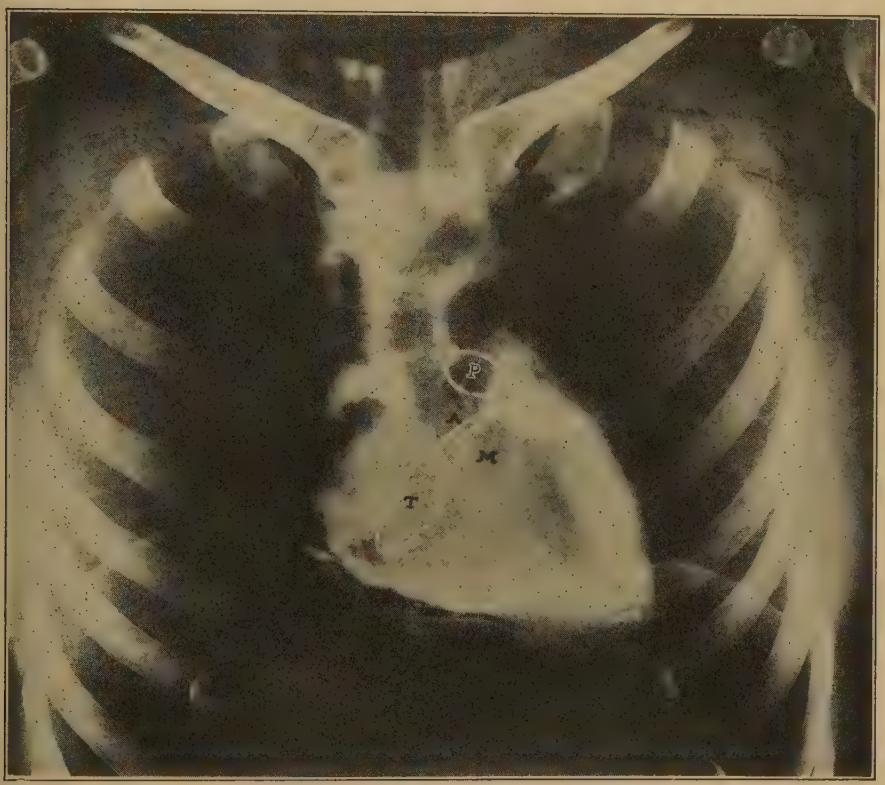



FIG. 16A.—Exact location of the heart, etc. See Legend on Photo. (*Courtesy of Dr. L. T. Le Wald.*)

given uniform results. According to Piersol, the aortic valves lie behind the left half of the sternum, a little below and to the right of the pulmonary valves; the latter are situated behind the sternal end of the third left costal cartilage. The tricuspids are situated behind the midsternum, opposite the fourth interspace and the fifth chondro-sternal articulation; the mitral valves are opposite the sternal end of the third left interspace (Figs. 16, 16A).

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## CHAPTER II

## FETAL CIRCULATION—CONGENITAL MALFORMATIONS, DEFECTS AND ANOMALIES

**Fetal Circulation.**—It will simplify the understanding of some of the congenital anomalies by recalling the salient features of the fetal circulation. The blood for the fetus is collected from the placenta by the umbilical vein. This vein enters the fetal abdomen through the umbilicus and courses along the suspensory ligaments of the liver to the under surface of that organ. Here the blood of the umbilical vein is divided into three channels: (1) the greater quantity passes by means of the hepatic veins through the liver with the portal venous blood before the latter enters the inferior vena cava; (2) some blood goes directly to the liver, to be also returned to the inferior vena cava by the junction of the ductus venosus and left hepatic vein; (3) a small quantity goes directly into the vena cava by the junction of the ductus venosus and left hepatic vein.

**Congenital Malformations, Defects and Anomalies.**—Although it had been formerly held that maternal impressions, injuries to the mother, and fetal endocarditis were the usual causes of congenital anomalies, malformations and defects, modern research has shown that practically all congenital defects are of developmental origin. Maternal impressions and injuries may now be entirely discarded as etiological factors. Regarding fetal endocarditis, an occasional rare case of sclerosis, especially of the pulmonary valve, may be exceptionally caused by a severe infection, particularly rheumatism, in the pregnant mother.

Developmental defects may be subdivided into: (1) those due to completely arrested developments; (2) abnormal developments; (3) abnormal septal deviations. All occur in early fetal life before the affected structures are fully grown. In a fair proportion of cases, other congenital anomalies, such as imperforate anus, absence of the fibula and complete or partial transposition of the viscera are found. Indeed, such more striking anomalies alone may direct attention to the heart as another possible congenitally defective organ.

It should be remarked that septal defects, complete absence of the septum and other anomalies do not often occur as "pure" cases; there is very frequently an admixture of anomalies in the same heart.

## SEPTAL DEFECTS

Although septal defects have been variously subdivided, it will be sufficient for our purposes to adopt the following simple classification to indicate the usual types.

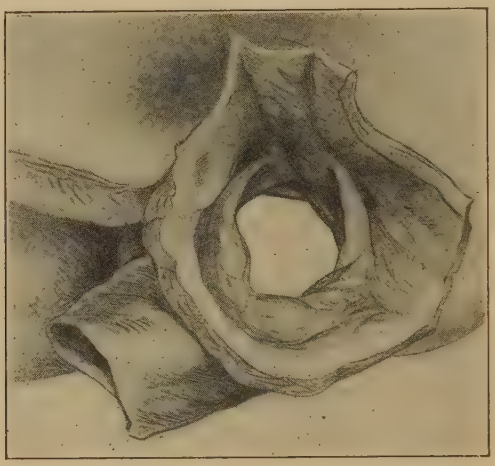

FIG. 17.—Patent foramen ovale with cornua of the embryonic valves still remaining apart. Pulmonary stenosis is also present. (*After Peacock.*)

**1. Defects of the Interauricular Septum. Patent Forman Ovale** (Fig. 17).—It is very common to find that the foramen ovale in the adult has not been entirely closed, leaving a small slit-like opening. Referring however to appreciable sized foramina, patent foramen ovale is not only by far the most frequent defect of the interauricular septum, but it is the most common of all congenital defects (191 out of 631 defect cases—Abbott). The size of the opening varies considerably, from one admitting an ordinary lead pencil to one several centimeters in diameter.

Other malformations of this septum consist in defects of its upper posterior, and of its lower portion. The latter are probably due to failure of closure of the *septum primum* (q. v. Chapter I). Another septal deformity consists of multiple foramina in the interauricular wall.

**2. Defects of the Interventricular Septum.**—These are not uncommon (189 out of 631 defect cases—Abbott). Almost all of these defects occur at the base of the septum (Fig. 18); rarely elsewhere. Such basal defects are most commonly combined with pulmonary stenosis and a right sided aorta. It has already been pointed out that in the embryo, the aortic septum is pushed downward to form part of the upper interventricular septum (q. v. Chapter I) at the *pars membranacea* (Figs. 11, 12). It would therefore appear that basal interventricular defects are probably due to arrest or non-development of the downward prolongation of this embryological aortic septum. In most of the defects of the interventricular septum,

it is rather surprising to note that the conduction system (Chapter I), although it lies in close proximity to the undefended space (*pars membrancea*) and hence to the basal defect, is little if at all disturbed in its course.

**3. Aneurism of the Undefended Space.**—Several observers (especially Rokitansky) have recorded instances of this interesting anomaly (Fig. 19). It has occasionally been found as the site of malignant endocarditis. It

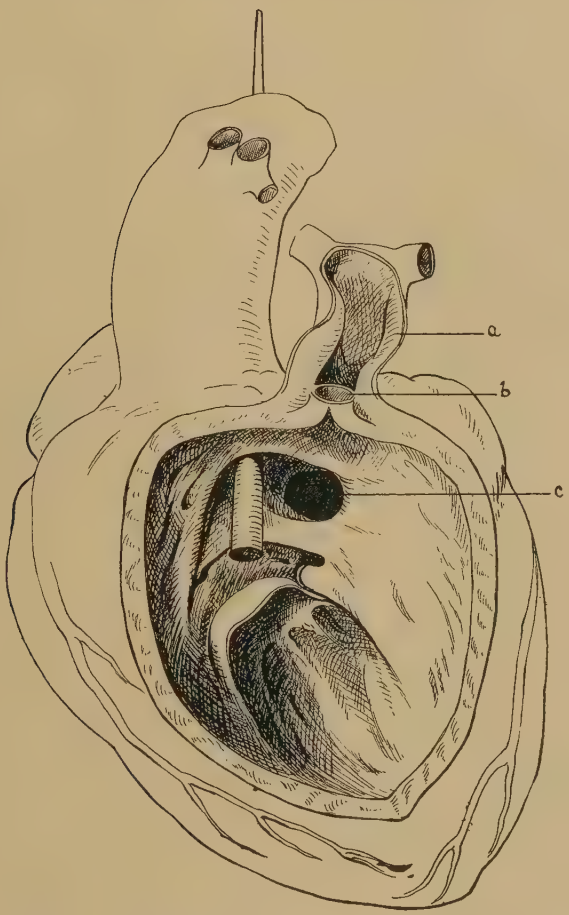


FIG. 18.—Patent interventricular septum. (*After Farre.*)

has therefore been held by some that endocarditis is the cause of such aneurisms. According to Mall, however, the cause lies in malposition of the inferior septum (q. v. Chapter I); as a consequence, the membraneous septum develops in a horizontal plane and thus becomes subject to undue mechanical strain and impact from the circulating blood, with the final result of the development of an aneurism.

**4. Gross Defects or Entire Absence of Interauricular and Interventricular Septa.**—Septal defects so large that only rudiments of the septa persist give rise to various types of two or three chambered (bi- and tri-loculated) hearts (*cor biloculare*, *cor triloculare*). There may co-exist atresia of the mitral or tricuspid valves, and, in addition, an abnormal origin or rudimentary development of either the aorta or pulmonary artery, or of both.

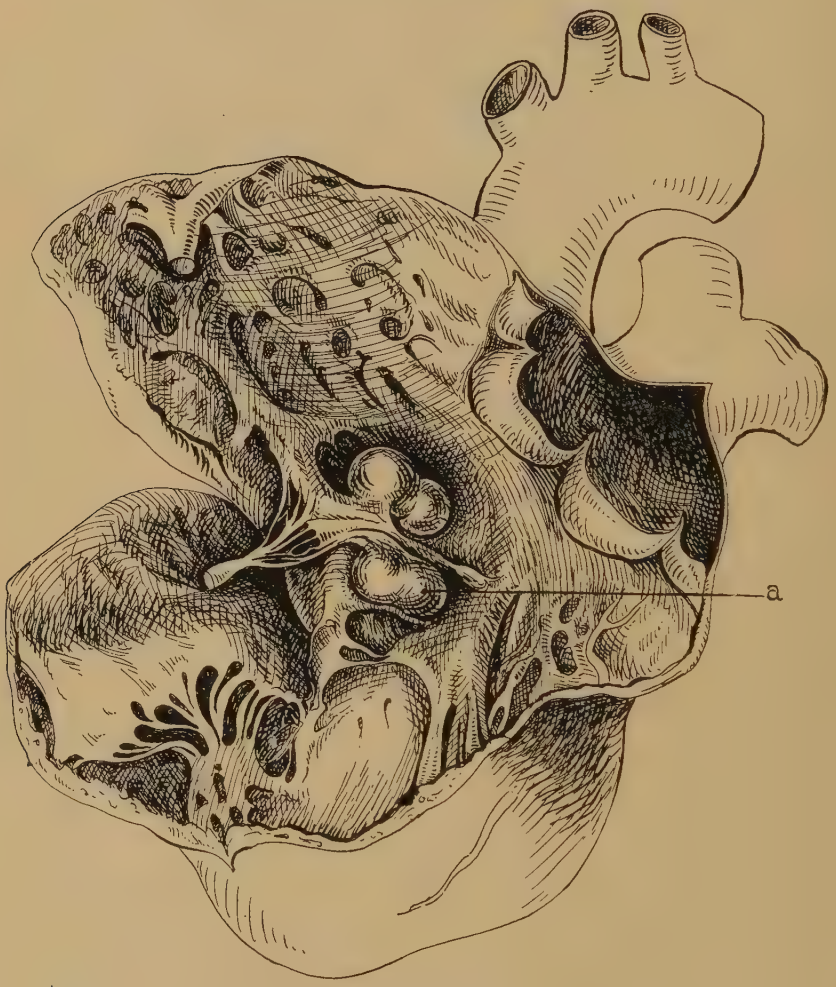


FIG. 19.—*a*, Aneurism of the undefended space. (*After Rokitansky.*)

A curious septal anomaly consists in the presence of an interauricular septum and the absence of an interventricular one, *i.e.* a three-chambered heart: two auricles with a common ventricle (*cor biatriatrum triloculare*). In rare instances there exists another type of triloculated heart: the inter-

auricular septum is absent and the interventricular one is present; there are thus two ventricles and a common auricle (*cor biventriculare triloculare*).

**5. Defects of the Septum of the Aorta and of the Truncus Arteriosus.**—The embryological formation of the aorta and pulmonary artery by the growth of the aortic septum has already been described (Chapter I). If there be entire absence of the aortic septum, there results a persistent *truncus arteriosus;* the latter, as the common arterial trunk, then usually receives the blood from both ventricles and gives off a pulmonary artery branch, in addition to supplying the usual branches of the aorta. A persistent *truncus arteriosus* is frequently combined with bi- or triloculated hearts, or with single interventricular defects.

**6. Valvular Stenoses and Atresias.**—There are varying degrees of congenital stenosis which may affect the cardiac valves. When there is complete obliteration of the canal, the condition is called atresia. Although most valvular stenoses and atresias are regarded as of developmental origin, some of the stenoses, especially where they form the only anomaly, are believed to be of inflammatory origin, the result of fetal endocarditis.

Stenosis or atresia of the ascending aorta is quite rare.

Stenosis and Atresia of the Pulmonary artery form alarge and very important class of the cardiac defects. Either anomaly may affect the vessel at its cardiac insertion (the conus), at the level of the valves, or, finally, the calibre of the artery beyond the valves. In rare instances a large part of the pulmonary artery from and including its insertion may be involved. A developmental triad that forms a common associated picture consists of pulmonary stenosis, defect at the base of the interventricular septum and a rightsided aorta receiving blood from both ventricles.

Atresia and stenosis of the auriculo-ventricular valves are exceedingly rare anomalies. To judge from associated pathological changes, some of the cases seem to be the result of fetal endocarditis, the others of developmental origin. Atresia and stenosis affect the tricuspid oftener than the mitral valve.

**7. Septal and Valvular Anomalies.**—Anomalous intra-auricular septa have been reported. Those occurring in the right auricle consist of a weblike reticulum stretching across the auricular chamber (Chiari). They are ascribed to mal-development of the *septum spurium* (q. v. Chapter I) or of the venous valves (Chiari). Cases have been reported in which the left auricle is divided into two chambers by the formation of a membrane; the latter is perforated so as to allow the blood from the upper chamber (into which the veins empty) to flow into the lower chamber and thence to the mitral orifice. This developmental anomaly is regarded as due to a misplaced *septum spurium* (Borst, quoted by Abbott).

The auriculo-ventricular valve may be duplicated by a similar supernumerary valvular apparatus with its papillary muscles, chordæ tendinæ and valve leaflets, thus forming a double auriculo-ventricular opening. Semi-

lunar cusps may be increased or decreased in number as the result of developmental defects; the largest number described has been five; the smallest two In the latter instance, it is probably the result of fusion in fetal life of the rudiments of the individual leaflets.

Occasionally fenestra of larger or smaller size occur in the semilunar valves (Aschoff).

So-called false tendons may traverse either ventricular cavity. It is now known that in the majority of cases these anomalous structures are not tendinous in nature but are large, coarse, aberrant ventricular branches of the auriculo-ventricular conduction system (q. v.).

**8. Defects and Anomalies of the Ductus Arteriosus (Ductus Botalli).**—As is known, the ductus arteriosus is a short, patent, fetal vessel connecting. the aorta and pulmonary artery. Normally it becomes impermeable within two or three weeks after birth. When patency persists—a comparatively frequent congenital anomaly—it is usually combined with pulmonary atresia or stenosis. The lumen of a patent ductus arteriosus varies considerably in size, from a scarcely perceptible opening to one admitting a pencil or even larger. Occasionally, the persistent duct becomes dilated. This condition has been called aneurism by some observers, but the name is apparently not justified, for no change in the vessel wall comparable to that of an actual aneurism has been found.

**9. Deviations and Transpositions of the Arterial Trunks.**—Rokitansky has described in great detail his theory of the causes of deviations and transpositions of the aorta and pulmonary artery. Briefly expressed, he attributes the phenomena fundamentally to varying degrees of congenital deviations of these vessels. He conceives the anomaly as a fault in the development of the aortic septum. If, for example, the concavity of the aortic septum be deviated so that it faces anteriorly, it would give rise to an *anterior* left aorta and a *posterior* right pulmonary artery. This exactly reverses the normal relationship of the aorta and pulmonary artery, for the pulmonary artery normally originates anterior to, and to the left of the aorta (Figs. 11, 12), and passes backward underneath the aortic arch. The normal aorta at its origin lies to the right of, and in a plane posterior to the root of the pulmonary artery.

By means of various deviations and transpositions of the fetal aortic septum in the primitive aortic bulb (Figs. 1, 2), combined with mal-union of the aortic and fetal interventricular septa, several types and combinations of anomalies can be readily conceived. For example, a deviated aortic septum (and with it, the aorta) could fuse with a normally placed or with a misplaced interventricular septum. In the former instance, the aorta and pulmonary alone are transposed; the aorta then surmounts the right ventricle, and the pulmonary artery, the left ventricle. The ventricles retain their normal positions. This represents complete transposition of the arterial trunks. With fusion of aortic and interventricular septa—both misplaced—there is cor-

responding transposition of position of right with left ventricle, so that the transposition of the vessels may be regarded as "corrected;" that is to say, the transposed (now right sided aorta) arises from a transposed (now right sided) "left" ventricle. Such transpositions are therefore classified as "corrected" transposed arterial trunks.

Other arterial transpositions consist of double implantation of aorta and pulmonary artery from a common ventricle, either right or left, with a normal or transposed relationship of pulmonary artery to aorta (Abbott).

The semilunar cusps in almost all instances of these transpositions show various deformities, such as being of unequal size or being bicuspid. The valves become rotated in accordance with the abnormal position taken by the arterial trunks; the degree of rotation and deviation may be determined by observing, for example, the position of the non-coronary cusp of the aorta.

Although the theory of Rokitansky as already given seems to cover the facts, it should be observed that Keith believes the various transpositions can be best accounted for by assuming a fetal atrophic process affecting one arterial trunk, and an expansion of the remaining vessel; the final effect would thus also bring about various abnormal relationships of the aorta to the pulmonary artery.

10. **Co-arctation of the Aorta.**—This, a comparatively frequent congenital anomaly, consists of various degrees of narrowing of the aorta near the insertion of the ductus Botalli. The narrowing may vary from slight constriction to complete obliteration. The area of the aorta affected is the isthmus (the portion lying between the *ductus arteriosus* and the origin of the subclavian artery), which in fetal life is normally of smaller calibre than the remainder of the aorta.

11. **Aortic hypoplasia** consists of congenital general narrowing of the entire aorta and its branches. The vessel walls are usually thinner and finer in texture than the normal. With this condition there may also be similar diminution in the size of the heart.

12. **Anomalies of the Aortic Arch.**—The aorta and its chief branches are subject to various anomalous positions. These include vicarious insertions and courses of the aortic branches. The fundamental reason for the anomaly lies in the fact that in fetal life, the aorta represents a double tube with six embryonic arches (Figs. 20, 21). Several of these normally disappear, while those that persist form the carotids, the aorta, the subclavians, and the pulmonary artery with the ductus arteriosus.

13. **Anomalies Affecting the Entire Heart.**—If the embryonic process affecting the heart be interfered with in early fetal life, the heart may be merely a rudimentary organ (hemicardia), or indeed there may be no cardiac organ at all (acardia). As an exceedingly rare finding in man, more than one heart may be present. The apex of the heart may be bifid, due to persistance of the fetal interventricular groove (q. v. Chapter I). Such a bifid apex may

occur as the only anomaly. The heart may be congenitally displaced outside of the thorax, either through a fissure in the sternum (*ectopia cordis pectoralis*) or through an opening in the diaphragm. In the latter instance, it becomes

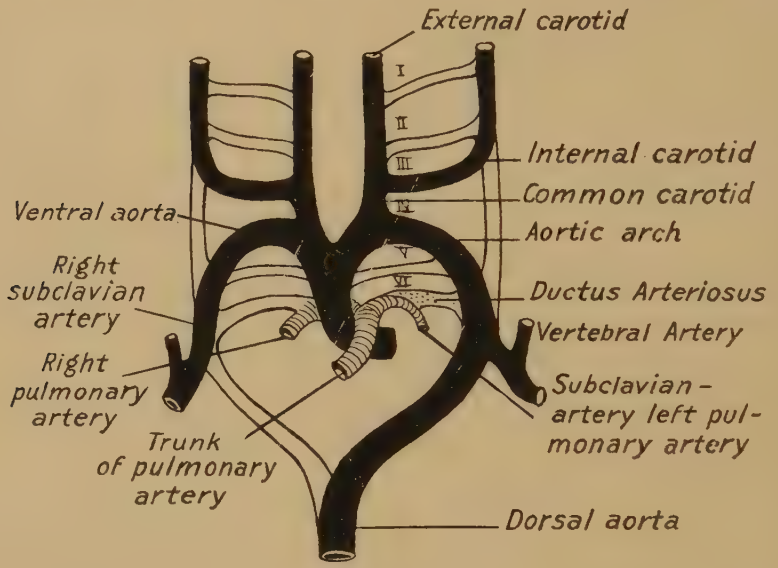


FIG. 20.—Diagram showing aortic arches and their derivatives in the human embryo. (*After Prentiss.*)

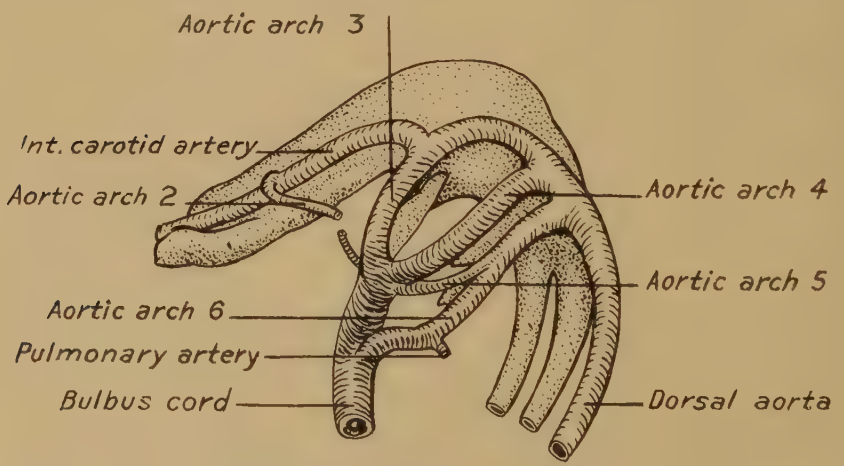


FIG. 21.—Aortic arches of human embryo of 7 mm. (*After Tandler.*)

an abdominal organ (*ectopia cordis abdominalis*); this condition is not necessarily incompatible with life.

14. **Congenital Dextrocardia.**—There may be no actual transposition but a simple vertical rotation in an anti-clockwise direction, so that the apex is

directed to the right, with the result that the left ventricle comes to lie more anteriorly, and the right ventricle more posteriorly than is normal. Most cases of congenital dextrocardia are of this type.

In another—a rare group—there is "mirror" transposition—(Abbott)—all rightsided structures become left, and vice versa. The apex, directed to the right, is formed, as normally by the left ventricle. Congenital dextrocardia is often accompanied by transposition of other viscera; for example, of the gastro-intestinal canal, the testes, liver, etc. The diagnosis of congenital dextrocardia by means of electrocardiograms will be discussed later in another connection (Chapter IX).

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## CHAPTER III

### PHYSIOLOGICAL CONSIDERATIONS—NORMAL SPREAD OF THE WAVE OF EXCITATION—THE EXTRINSIC CARDIAC NERVES—THE CARDIO-INHIBITORY CENTER—MYOGENIC OR NEUROGENIC IMPULSES?

It has long been known that certain fluids appropriately introduced into the isolated frog's heart will keep that organ beating for a long time; such artificial circulation consists essentially in a method of perfusing the heart by introducing the fluid through the veins. Blood and lymph were originally used. Later, various solutions were employed, and their effect upon the contractility of the isolated frog's heart was studied. Ringer discovered that a perfusion solution containing a mixture of sodium, potassium and calcium chlorid would keep a frog's heart beating for many hours. An effective Ringer's solution for experimentation purposes upon the frog or turtle heart should contain 0.7 per cent. sodium chlorid, 0.03 per cent. potassium chlorid and 0.025 per cent. calcium chlorid (Howell). A physiological salt solution alone (sodium chlorid, 0.7 per cent.), if perfused, produces rhythmical contractions for a comparatively short time; the contractions gradually become weaker and finally stop. In order to keep an isolated mammalian heart alive, Locke's solution is preferable; it contains sodium chlorid, 0.024 per cent.; potassium chlorid, 0.042 per cent.; bicarbonate of soda, 0.01 to 0.03 per cent., and dextrose 0.1 per cent. The dextrose, while not essential, increases the effectiveness of the mixture. The mammalian heart is perfused under an atmosphere of oxygen.

The results of much experimentation have shown that, in order to maintain cardiac contractility of the isolated heart most efficiently, the salts of sodium, potassium and calcium must be employed, and these in certain definite proportions. If, for example, a turtle's heart be perfused with blood serum from which the calcium salts have been removed by precipitation with sodium oxalate, the heart comes to a standstill, to again contract normally when the proper proportion of calcium chlorid has been added to the oxalated blood. While it is evident that the ions just mentioned have an intimate and necessary connection with the maintenance of cardiac contractility, and while it seems probable that each metal has an individual and specialized function relating to cardiac systole and diastole, it is as yet unknown what their definite correlation is to the contractile substance of the heart or to the fundamental "inner stimulus"—be it chemical, neurogenic or otherwise—which produces the heart beat (Howell). On the whole, it seems probable that the calcium, sodium and potassium salts act antagonistically, that the

calcium promotes contractility, while sodium and potassium salts produce a state of relaxation.

Engelmann and Gaskell, by careful experimentation about forty years ago, established certain attributes of the cardiac musculature, namely, irritability, contractility, rhythmicity and conductivity. These have been respectively designated by Engelmann as bathmotropic, inotropic, chronotropic, and dromotropic functions; he has qualified them as positive or negative, depending upon influences which act favorably or unfavorably upon the individual functions. This nomenclature, however, has not found its way into the general literature of cardiology. To the four attributes of cardiac musculature, Gaskell has added a fifth—tonicity. As will be seen later, various portions of the heart are differently endowed with these five special attributes.

**Rhythmicity.**—Thorough experimental and electrocardiographic investigations in mammals, and histological and pathological studies in man and animals, have proven, as already stated (Chapter I), that the *primum movens* —the center for rhythmicity—the normal impulse center in the human being —exists in what is variously known as the pacemaker, the sino-auricular node (the S-A node), or the node of Keith-Flack. The classical ligature experiment of Stannius (first ligature of Stannius) many years ago proved that in cold blooded animals, the rhythm center is situated in the region of the *sinus venosus*. He ligated the heart in the sinus region: The auricle and ventricle ceased beating; the sinus continued to beat. Other more recent corroborative evidence that the sino-auricular node is the normal pacemaker is the fact that experimental excision of the node usually causes the auricle to stop beating entirely or to beat very slowly; and, further, the electrocardiographic observation that the node becomes electrically negative (that is, it shows activity) before the remainder of the sinus region.

While the node is the normal pacemaker, it is well to emphasize here that clinical conditions occur in which the normal pacemaker does not act but that other portions of the heart—usually other portions of the auricle—take up the pacemaking function, and become for the time being the rhythm center. These abnormal clinical conditions will be described later (Chapters X, XI).

As the result then, of all these different investigations, it may be definitely stated that the normal excitation processes, the normal center from which impulses in the human heart start, originate in the pacemaker, the sino-auricular node.

**Conductivity.**—This property of the cardiac musculature deals with the manner in which impulses starting from the pacemaker spread throughout the remainder of the heart muscle, that is, through the auricles and ventricles. In these observations of the respectively excited portions of the cardiac musculature, use has been made almost entirely of electrocardiographic tracings. These will be described in detail in their proper connections

(Chapters VIII–X). For the better understanding of the spread of the excitation wave, it will suffice to state at this time that every muscle in contracting produces a small amount of electricity; that this electricity can be conducted (led off) from the contracting muscle by means of non-polarizable electrodes. In animal experiments such electrodes can be placed directly in contact with the various portions of the heart whose excitation and contracting processes one wishes to study: This constitutes the direct method of electrocardiography (Figs. 59–69). For other purposes the indirect method is employed. It consists of different types of non-polarizable electrodes which are placed around each forelimb and left leg of the animal, corresponding to the electrodes placed upon the forearms and left leg (Figs. 64, 65) when a human electrocardiogram is taken. In either case, the muscle (Figs. 59, 60) (in this instance, the heart) becomes electro-negative during contraction; indeed, electro-negativity and muscular contractility are synonymous and interchangeable terms. This wave of electric excitation is led off by means of wires which connect the electrodes to the electrocardiographic apparatus (Figs. 59–69) with its "string"—a very fine silvered quartz fiber. The string becomes activated and moves as the result of the electrical excitation produced by cardiac contraction. The movements of the string are magnified and photographed (Figs. 67, 69). The resultant picture constitutes the electrocardiogram.

In order to study the paths taken by impulses arising from the sino-auricular node, non-polarizable electrodes were placed in contact with various appropriate parts of the cardiac musculature, and those portions which first became electrically negative (and therefore which first contracted) as compared with other parts, were recorded by means of electrocardiograms. In this manner it was demonstrated that the excitation wave spreads ripple-like over the auricles from the sinus node as a center at a rate of about 1000 millimeters per second. The excitation wave follows the chief auricular muscle bands which radiate from the sinus region, and spreads thus throughout the auricular musculature and auricular septum to reach the junctional tissue (the auriculo-ventricular or A-V conduction system (Chapter I).

After the excitation impulse reaches the A-V node, its spread through the ventricles does not follow the anatomical arrangement of that musculature (Chapter I). From the A-V node, the excitation wave first spreads almost simultaneously to those areas supplied most directly by the right branch of the A-V bundle, that is, the part of the right ventricular wall which lies over the large anterior papillary muscle; later, the remainder of the right ventricle becomes excited and contracts. In the left ventricle the wave also follows a definite course. The earliest point to be excited is the muscular vortex of the left ventricle near its apex or the extreme apex itself; about one hundredth of a second later the neighboring points are activated; excitation of the remainder of the left ventricle is almost simultaneous. The cardiac base and the region of the conus are usually the last to receive the excitation

impulse. Since some areas of the ventricular musculature are excited practically simultaneously, the ventricular wall must presumably be reached by simultaneous impulses traveling along a large number of paths; by experiments, these have been shown to be the Purkinje fibers (Chapter I). But some parts of the ventricular musculature not supplied by free, branching Purkinje strands (*e.g.* the conus region beneath the pulmonary valves) are activated very early. This has been demonstrated experimentally to be due to the thinness of the ventricular musculature in such regions. In other words, the excitation wave is conceived as traveling not only along the surface of the interior of the ventricular chambers but also as piercing the musculature. The varying thickness of the musculature also explains the reason for the excitation wave reaching the thinnest area first and later the thicker left ventricular areas. Excitation is found to travel extremely rapidly through the conduction system; the rate is about 5000 mm. per second. Through the muscle itself, however, it is much slower, namely about 500 mm. per second. In brief, then, the excitation wave appears almost simultaneously along the interior of both ventricles. The activation of the ventricular surface is only partly dependent upon its distance from the Purkinje fibers; it is mainly controlled by the time required for the excitation wave to pierce the thicker musculature overlying the latter fibers.

**To Summarize.**—From a study of the physiological and anatomical distribution of the specialized tissues, as well as from experimental electrocardiographic investigations, it is evident that the normal impulse arises in the S-A node; it spreads thence through the auricles, following, as far as known, no especially differentiated path in the latter. It then reaches the junctional tissue (that is, the A-V conduction system), and by way of its right and left branches and its terminal aborizations (the Purkinje system), the normal impulse spreads to the papillary muscles and throughout the entire ventricular musculature.

**Contractility.**—A characteristic of the systolic contraction of the heart muscle which distinguishes it from skeletal musculature is that each systole is of maximum intensity. This property—the so-called all or none reaction—means that the heart muscle answers any stimulus sufficient to cause any response at all by a maximal contraction; that is to say, any stimulus produces either a maximal contraction or none at all. As is well known the contractility of skeletal and plain muscle varies with the degree of the stimulation, a law which is entirely different from that applying to the heart. Cardiac contractility therefore does not depend upon the strength of the impulse. It does vary considerably, however, with the state of irritability (q. v.) of the heart muscle, so that despite the fact that artificial stimulation of the mammalian heart produces maximal contraction, the resultant systoles are by no means necessarily alike, for the state of irritability of cardiac muscle may vary considerably, depending for example, upon the state of nutrition of the muscle at the time of stimulation.

This so-called "all or none law" or law of maximal contraction of cardiac muscle seems to depend upon the difference in the histological structure between skeletal and cardiac fibers. In the former, muscle bundles are encased in, and are separated by, sarcolemmal sheaths. In cardiac musculature such sheaths are absent. Hence stimulation of skeletal muscle may vary and produce sub-maximal contractions because all the fibers cannot participate at one time; in the case of the heart however, the whole muscle can, so to speak, contract as one fiber because of its histological continuity. Furthermore, the intricate intertwined muscle layers of the heart, already described (Chapter I) also tends to make the heart act physically as one continuous muscle.

**Irritability.**—The heart is not irritable during the entire time of its systole, that is to say, if an electric stimulus be applied to the heart during its stage of contraction, it has no effect upon the contraction. This is termed the refractory period of the heart beat. In consequence of this, the heart is irritable only during its diastolic or resting period. A stimulus applied during diastole produces an extra or premature contraction, called technically an extrasystole (q. v.). The heart therefore is different from skeletal musculature in that it cannot be tetanized like the latter. There is an experimental exception to this law; hearts that have been poisoned by muscarine, alcohol, chloral, etc., may have a shortened refractory phase and hence may be brought into tetanic contraction by proper stimuli.

The irritability of the heart returns gradually during its diastolic period. It would therefore seem that the return of cardiac irritability is intimately associated with some as yet unknown metabolic change or changes going on in the cardiac musculature.

**Tonicity or Tonus.**—This property is similar to that found in ordinary skeletal musculature, which, in the case of the heart, keeps it in a slight state of contracture even during diastole. Tonicity is a term which is perhaps used too loosely and indiscriminately, and is often confused with the contractile power of the heart. Tonicity is the attribute about which few clinical or experimental facts are known. It is apparently intimately connected with various degrees of cardiac dilatability—an important clinical complex—yet tonicity is in so far an individual property in that it may be present even when the heart is dilated. Furthermore the degree of tonicity does not necessarily vary proportionately with the amount of cardiac dilatation. It would thus seem that tonicity is a property apart from contractility. This has received some verification from Porter's experiments. These demonstrated that tonus contractions vary with the strength of the stimulus and have no refractory period, and that tonus tetanus can be produced by proper stimuli. As has been pointed out, the characteristics of cardiac contractility are just the reverse, for the heart muscle has a refractory period, contraction does not vary with the strength of the stimuli and heart muscle cannot ordinarily be tetanized.

**The Physiological Action and Influences of Cardiac Nerves.**—The anatomical distribution of the cardiac nerves and the relationship of the vagi and sympathetics to the conduction system—the sino-auricular node and the bundle of His—have already been described (q. v.). The heart is often profoundly influenced by impulses which reach it from these nerves. The vagi contain the inhibitory fibers. If, in the experimental animal, the vagi be cut in the neck, the cardiac rate will be increased. If the peripheral ends of the cut vagi be stimulated, there will be slowing or stoppage of the heart, or a condition in which the auricles beat more rapidly than the ventricles, with no rhythmic relationship between the two (complete heart block) (Chapters X and XX). In warm-blooded animals there is not only a diminution of cardiac rate, but also of the strength of auricular and ventricular contractions, until finally the heart stops in diastole. On the other hand, stimulation of the cut end of the sympathetic produces a varying degree of cardiac acceleration. Depending upon the degree of stimulation of vagus and sympathetic, the one or other nerve has a predominant influence upon the cardiac rate. Although antagonistic, this antagonism cannot be measured purely arithmetically for if vagus influence predominates during the course of the experiment, with cessation of stimulation a typical accelerator influence results. This fact in itself indicates that a certain amount of "tone" is present in both nerves. Nerves of sensation have not been discovered in the heart. However, since the lower cervical and upper dorsal nerves supplying the integument of the neck, chest and upper extremity also send filaments to the deep and superficial cardiac plexuses, painful skin areas of varying degrees of intensity and extent are found; such painful areas are the result of reflex excitation originating in some pathological process or function of the heart itself.

As stated, stimulation of the vagus sometimes has as its most marked effect not slowing of the pulse rate, but diminution of the strength of the auricular and ventricular contractions. The latter, for example, may be considerably weakened and lessened in amplitude. Vagus stimulation, however, seems to have a more marked influence upon auricular than upon ventricular systoles. Indeed, it is questionable whether the effect upon the ventricle is not secondarily caused by the inhibitory action of the vagus on the sinus region, particularly upon the sino-auricular node (the pacemaker), thus causing inhibition of normal impulses passing from auricle to ventricle. As an occasional result of vagus stimulation the ventricles may either continue beating or may stop entirely when the auricles are brought to a standstill. The cause for these occasional variations is not clear; they may possibly be due to varied distribution of branches of the vagi to the sino-auricular and atrio-ventricular nodes (Chapter I), or to differences in vagal and accelerator nerve tone. Other reasons adduced for these occasional variations of ventricular action following vagal stimulation are increased ventricular diastole produced by slowing of the heart rate, and differences in the strength of the

stimuli applied in the various experiments (Tigerstadt). Despite these contradictions it seems probable that the vagus has a decided influence, directly or indirectly, on cardiac rate and contractility, as well as on conductivity.

Because of dissimilar distribution of the right and left vagus nerves (Chapter I), it has been shown experimentally in the dog's heart that stimulation of the right vagus usually brings the heart to a standstill: that, because of its predominant distribution to the auriculo-ventricular conduction system, stimulation of the left vagus influences and interferes with conduction from auricle to ventricle. Such interference may vary from simple prolonged conduction time from auricle to ventricle (Chapters X, XI), to incomplete heart block (Chapters X, XI); or in more marked instances it may end in a condition in which the auricles beat but no impulses pass to the ventricles; hence the latter finally cease beating (A. E. Cohn). These experimental observations have definite clinical application, especially in attempting to control paroxysmal tachycardia by pressure over the vagus in the carotid sheath (Chapter XI).

The primary effect of experimental stimulation of the accelerator is an increase of cardiac rate; this increase may be slight or marked. Combined with the acceleration there may or may not be increased amplitude of the cardiac contractions. Because of such possible differences in amplitude, some physiologists believe that there are augmentor as well as accelerator fibers in the accelerator nerve. There seems to be a distribution of right and left accelerators to the sino-auricular and auriculo-ventricular nodes comparable to that of the vagi, that is, the right accelerator supplies predominantly the sino-auricular, and the left accelerator, the auriculo-ventricular node.

Regarding the ganglia, stimulation of the right stellate ganglion causes an increased auricular and ventricular rate, but no change in conduction time. Stimulation of the left stellate ganglion produces similar acceleration, in addition to shortening of the auriculo-ventricular conduction time (Rothberger and Winterberg); this suggests increased irritability of the auriculo-ventricular node and bundle of His, probably corresponding to a preponderant nerve supply from the left accelerator.

**The Cardio-inhibitory and Accelerator Centers—Reflex Excitation.**—The location of the accelerator center—whether in the medulla or elsewhere—has not been accurately defined. It is known that the center must be in the brain, for stimulation of the cervical region of the spinal cord produces acceleration. As for the cardio-inhibitory center, it is definitely known to be in the medulla (Figs. 14, 15).

There is much clinical and experimental evidence that the cardio-inhibitory center as well as the vagi themselves can be reflexly stimulated and influenced in many ways. Thus centripetal (afferent) nerves from the skin, from the heart itself, from the gastro-intestinal canal, lungs and the special

sense organs can affect the cardio-inhibitory center. In addition, such central influences as sorrow and joy can have a decided action upon the cardiac rate.

The classical example of reflex inhibition is that of Goltz who, by continued light tapping upon a frog's abdomen, brought the heart to a standstill: with the vagi out, cardiac standstill was not obtained.

In man, the afferent (centripetal) impulses—whether from skin, muscle or organ.—are carried by means of the central nervous system to the cardio-inhibitory center in the medulla; from there the efferent (centrifugal) inhibitory impulses pass along the vagi. In addition, the center in the medulla may be directly affected by pressure of new growths or by neighboring inflammatory reactions, and especially by changes in the composition of the blood supply of the center. It is important to recall that, as with other centers, the cardio-inhibitory when influenced by afferent impulses, may be either excited or inhibited. When inhibited the cardiac rate is accelerated. If when excited the center be already active, then the cardiac rate is retarded.

**Myogenic or Neurogenic Impulse?**—I have already indicated the probable course of the normal impulse in the mammalian heart and have also emphasized the rich nerve supply of the sino-auricular node and of the auriculo-ventricular conduction system. These facts lend renewed interest to the old question in dispute for many years, especially among physiologists, whether the "inner stimulus" is of neurogenic or myogenic origin. It will perhaps be advisable to set forth the salient features of the myogenic theory (at present the more popular among physiologists), and to give the objections thereto from the standpoint of the neurogenic theory.

1. Those favoring the myogenic theory point out that the heart of the embryo chick pulsates before it contains nerve cells (His, Jr.). This is one of the strong basic observations upon which the myogenic theory is founded. The antagonists state that it is open to the usual objections of negative findings (Cyon), for with better technique and further progress in neuro-embryology, nerve structure may be discovered in the early embryonic chick's heart.

2. Haphazard strips cut from hearts of coldblooded animals, when placed in proper solutions, will continue to beat. This argues for inherent rhythmicity in the muscle fiber, for it does not appear probable that every bit of cardiac tissue thus haphazardly cut off has its own arrangement of nerve tissue acting as a motor center. The neurogenic theory holds that such nerve structures as are found in the isolated strips of cardiac muscle tissue are sufficient to induce rhythmic contractions.

3. If a zig-zag preparation of the frog's heart be cut so that there are in places only slight, frail muscular bridges (Engelman), and the preparation be stimulated at any point, the whole muscle preparation will contract. The objection offered by the neurogenists to this experiment is that there are innumerable nerve anastomoses along which the excitation wave may travel.

4. The discovery of the sino-auricular node, and the propagation to the ventricles of impulses by muscular continuity (the bundle of His, its two chief branches, and their aborizations) argues strongly for a primary myogenic impulse. The counter-argument (Cyon) is that it does not seem probable that so small a muscular bridge can carry all the impulses from auricle to ventricle.

5. Section of the auriculo-ventricular bundle, by breaking muscular continuity, interferes with conduction of impulses and produces heart block (Chapters X, XI), that is, dissociation between normal auricular and ventricular contraction. The counter argument is that the bundle also contains nerve structures which are likewise demolished by the experimental section of the bundle, hence the interference with conductivity.

6. Isolated heart muscle cells of a chick embryo, placed in a plasma medium, may be kept alive and continue to beat (Burrows). The answer to this argument is that even in the absence of nerve cells, an embryonic function, originally muscular, need not necessarily continue so. For example, Carlson has shown that in the heart of the limulus, the embryonic impulse, originally muscular, later becomes neurogenic.

The intimate and intricate relationship existing between the muscle and nerve structure in the nodal regions demonstrates how difficult must be the final determination of this question of neurogenic and myogenic impulse. It is known clinically that the rhythm center is readily influenced by purely neurogenic impulses; these can indeed upset the normal cardiac control and give rise to abnormal rhythms. How far such clinical observations can be applied to the question of the normal control of the rhythm center it is impossible to state. All in all, it seems probable that the sino-auricular node is activated by both neurogenic and myogenic influences, although we have at present no means of discovering under which circumstances the one or the other becomes the controlling factor.

Whether myogenic or neurogenic, however, the important question to decide is the primary nature of the automatic, intrinsic stimulus (the "inner" stimulus) which fundamentally excites the heart beat. The prevailing opinion at present is that its cause lies in the chemical composition of the blood, particularly in the sodium, calcium and potassium content of the plasma.

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## CHAPTER IV

## CARDIAC HISTOLOGY AND PATHOLOGY

The structure, distribution and histology of the sino-auricular node and of the junctional tissue have already been described (Chapter I).

The heart is composed of three layers: The endocardium, the epicardium and the myocardium.

The endocardium, the serous membrane of the heart, lines all the cardiac chambers and both sides of all the valves. It is continuous with the intima of the veins and arteries at the various cardiac orifices and is composed of two layers: A superficial single layer of endothelial cells; a deeper layer of loose areolar tissue containing fibrous and elastic tissue, and some smooth muscle cells. This deeper layer is continuous and merges with the intermuscular connective tissue septa of the myocardium, and is intimately attached to the latter. Strong fibrous rings (*annuli fibrosi*) consisting of fibrous and elastic tissue line the auriculo-ventricular openings. Rings of similar structure surround the openings of the aorta and pulmonary artery. The cardiac valves are composed of fibrous and elastic connective tissue covered by endocardium; they contain muscle fibers at their attached margins, which in turn are attached to the *annuli fibrosi*. The ventricular valves—the mitral and tricuspid—are thinner than the aortic and pulmonary valves.

The epicardium forms the visceral layer of the pericardium. It is composed of connective tissue, with comparatively few elastic fibers or fat cells, and of a single layer of squamous epithelium. It is closely applied to the underlying myocardium or subpericardial fat.

The pericardium is the closed sac in which the heart is suspended. It is a fibro-serous structure. Its inner visceral layer is the epicardium. The parietal layer contains much elastic and connective tissue, and is rich in lymphatics, nerves and blood vessels. At certain parts it becomes thicker and tougher so as to form pericardial ligaments. These ligamentous prolongations extend and become attached to the aorta and pulmonary artery at the base of the heart; to the upper and lower portions of the sternum; to the diaphragm near the inferior cava; and to the vertebra by merging with a mediastinal extension of the deep cervical fascia. The pericardial ligaments are mechanically important because they help to fix various portions of the heart during its systolic contractions.

The myocardium—the muscular layer of the heart—is made up histologically of a special type of involuntary muscle cell (Fig. 22) not found elsewhere in the body. The cells are striated and anastomose by means of short lateral

processes. Each heart muscle cell usually contains but one nucleus, centrally placed. A cell membrane, the sarcolemma, if at all present, is extremely delicate. The cells contain a large amount of sarcoplasm. The striations of heart muscle are finer and more delicate than those of skeletal musculature. The longitudinal striations represent fibrillæ in the sarcoplasm. The transverse striæ indicate light and dark discs. Through the middle of the former is found the so-called Krause membrane. Of special histological interest are intercollated discs or cement lines of irregular length which may or may not completely transect the fiber. Seen in longitudinal section, they appear as dark lines running transversely across the main fibers and side branches. The importance and significance of these discs or cement lines lies in the question as to whether they do or do not represent actual cell boundaries; for if they do, it would clinch the fact that cardiac musculature is composed of separate cells, and not as some suppose, of synticium—a structure in which there are no actual cell boundaries. Views regarding the interpretation of the discs are still at variance. The most recent interpretation is that the discs do not represent actual cell boundaries, and hence that heart muscle is not a cellular structure. The blood vessels, veins and lymphatics of the heart have already been described (Chapter I).

FIG. 22.—Muscle cells from the human heart showing longitudinal and transverse striations, and lines of union between cells. × 500. (*After Bailey.*)

## PATHOLOGY OF THE ENDOCARDIUM AND MYOCARDIUM—CARDIOSCLEROSIS

**Degenerative Changes of the Endocardium.** The endocardium is subject to the following degenerative changes:

**Fatty Degeneration** (Fig. 23).—Macroscopically, pure white or yellowish white areas are found in the endocardium, especially on the mitral valves in old people. Such degenerative areas usually represent retrogressive senile changes; they are, however, occasionally the result of anemia, toxemia and infections. The connective tissue cells in the degenerated areas contain more or less numerous large and small fat droplets.

Amyloid degeneration of the endocardium occasionally occurs. It is rarely the predominant pathological change.

**Sclerosis.**—The affected endocardium takes on a white, thickened, hyaline appearance. An entire valve (especially the mitral), or its free border, or parts

of the mural endocardium may be involved; in addition to these changes there may be calcareous deposits. Occasionally the sclerotic areas undergo mucoid degeneration. Microscopically, the sclerotic patches are found to consist of strands of connective tissue which have undergone hyaline degeneration.

**Atheromatous Degeneration** (Fig. 24).—This consists of focal necrosis in the valvular tissue and may include any of the changes already mentioned.

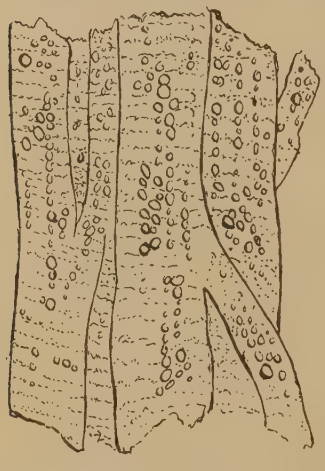

FIG. 23.—Fatty degeneration of heart muscle. (*After Kaufman.*)

The above degenerative types (sometimes called arteriosclerotic endocarditis when sclerosis is marked) are as a rule found in elderly people, and may be regarded as senile or retrogressive in nature. They are occasionally encountered in the middle aged, very rarely in the young. The valves especially attacked by these degenerative processes are those previously inflamed. The result is that the free surface of the valvular endocardium becomes roughened; adherent thrombi may form which may later become partially or totally organized as the result of a slow productive inflammation. Thickened patches are thus formed. The end result of all these changes is an irregular, more or less diffuse, sclerotic and atheromatous thickening of the valves, with consequent profound change in their structure, shape and function. They can no longer close the cardiac orifices properly, thus giving rise to various abnormal blood currents and consequently to murmurs (Chapter XIII). The arteriosclerotic type of endocarditis is especially apt to involve the aortic valves, usually at their bases. It is at times difficult to differentiate this primary sclerosis and atheroma from chronic fibrous and infectious endocarditis.

**Inflammation of the Endocardium—Endocarditis. Acute Simple Endocarditis.**—It is now generally believed that all types of acute endocarditis—rheumatic, mycotic, etc. are due to bacteria or to their toxins. Since bacteria have not yet been demonstrated in the blood of patients with simple, acute rheumatic endocarditis, the term simple acute endocarditis will here be applied to the pathological changes in the endocardium accompanying the latter condition. However, even this limitation is artificial for, as will be shown, the simple may be combined with other more destructive changes. Acute endocarditis is also termed verrucous, productive or rheumatic endocarditis. The valves are usually affected along their free borders of closure. According to our present knowledge these portions of the valves do not possess blood vessels, hence the assumption is that the valves are damaged by bacteria or bacterial toxins flowing in the blood stream. The damage is

FIG. 24.—Atheromatous degeneration of the aorta due to syphilis. (*Courtesy of Dr. J. Larkin.*)

occasionally so mild and superficial that the only sign of a previous inflammation is a small amount of scar tissue. The only pathological change may be simple swelling of the valves with tumefaction of the basement substance; the valve surfaces then retain their glistening appearance, there is only slight production of new connective tissue. In more severe cases, the growth

FIG. 25.—Ulcerative endocarditis showing large vegetations (V). (*Courtesy of Dr. J. Larkin.*)

of new connective tissue becomes the predominant feature. The basement substance splits, making way for fungoid projections of connective tissue masses. These are the so-called vegetations, hence also the name verrucous endocarditis. If cluster and deposits of fibrin adhere to the vegetations, the latter may reach enormous proportions. In other instances, production of new tissue and degeneration with ulceration go on simultaneously in different parts of the affected valve; the result—vegetation and loss of substance—is

characteristic of what is termed simple acute ulcerative endocarditis (Fig. 15). Since the verrucous and ulcerative varieties of endocarditis are not always distinct and do indeed merge at times, both may occur together as a mixed process. A shrinkage of this granulomatous connective tissue produces what is known as chronic fibrosis or fibroplastic endocarditis.

FIG. 26.—Malignant endocarditis affecting the aortic valves. V = vegetations. (*Courtesy of Dr. J. Larkin.*)

As shown by autopsies, these varying types or degrees of simple endocarditis—including even the simple ulcerative form—may occasionally regress so completely that there is scarcely any, or indeed no resultant valvu-

lar deformity. In such fortunate cases there is full recovery and restoration of all cardiac functions. In other instances the valves remain permanently damaged.

Acute endocarditis, either by recrudescence or as a continuing non-quiescent inflammatory lesion, may develop into chronic endocarditis.

**Acute Mycotic or Malignant Endocarditis** (Fig. 26).—Malignant, mycotic, ulcerative and septic endocarditis are the various names for that type of acute endocarditis in which the infective exciting agent is a bacterial organism recoverable from the blood. In the autopsy specimen the infective bacteria in large quantities can often be recovered from the damaged valve.

Malignant ulcerative endocarditis may occur as a primary process but it is usually an accompaniment of acute infectious diseases, or of pyemia.

The common sequence of the pathological process affecting the valves is as follows: The bacteria form clumplike deposits which appear as very fine grayish granules. Beneath these, there is destruction and necrosis of the endothelial, and finally of the deeper layers of the valvular structure. The extent of the destructive process apparently depends on the virulence of the invading bacteria. If the endothelial layer is not entirely destroyed, it shows only cloudy swelling. Thrombi from the circulating blood, consisting of platelets, fibrin, and white and red blood cells become adherent to the damaged surface. With the washing off of the bacterial clumps by the blood stream, there remains a small defect with a necrotic base to which thrombi again become attached. Where blood vessels are present in the valves at their bases, an exudative inflammation surrounds the necrotic areas. Where the vascular supply is absent there is a growth of connective tissue cells and an increase in lymphocytes; later, there may be formation of new blood vessels. In widespread, progressive mycotic disease, large reddish or dirty gray thrombotic masses may occupy large areas of the infected valves. The process may finally result in perforation of the valve and even of the *chordæ tendinæ*, with consequent production of loud, musical murmurs. The impingeing blood is also apt to break and carry off small friable masses, causing septic infarcts and abscesses in the lungs, kidneys and other viscera.

**Subacute Bacterial Endocarditis.**—This may be regarded as a special type of mycotic endocarditis, longer in its course than the acute variety and caused chiefly by the streptococcus viridans, exceptionally by the influenza bacillus. The pathological picture is practically that of mycotic endocarditis. For many reasons, the clinical picture of subacute bacterial endocarditis is of great importance and interest (Chapter XV).

**Chronic Endocarditis.**—As in the verrucous form, there are two distinct pathological processes found either separately or in combination.

The mural endocardium, but especially the valves of the left side of the heart, become thickened, rigid and covered with small, firm vegetations and infiltrated with lime salts. Or there may be simultaneous connective tissue growth and degeneration with ulceration and fibrin deposition on the

ulcerated roughened surface; shrinkage and lime salt deposits finally result (Fig. 27). The mural myocardium may also be affected by the spread and infiltration of the endocardial process.

Verrucous endocarditis is usually found in the left heart in adults. The ulcerative type is seen fairly frequently in the right side of the heart. In the new born, the site of predilection for verrucous endocarditis is also the right heart.

The verrucæ and the formation of fibrous tissue give rise to stenosis of the valvular orifices (stenotic lesion); or the valves no longer properly close

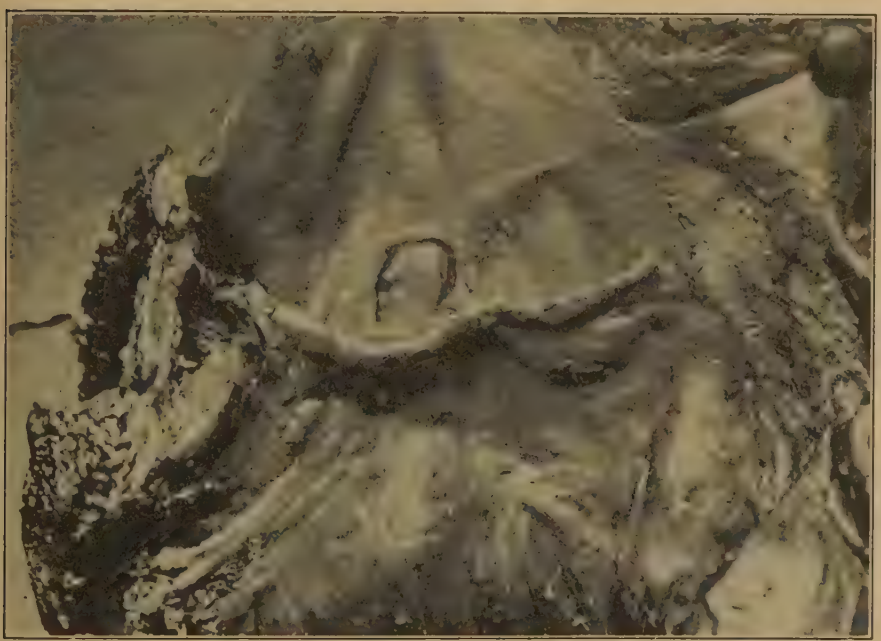

FIG. 27.—Chronic endocarditis with calcification of the thickened valves. Aortic insufficiency. (*Courtesy of Dr. J. Larkin.*)

the orifices, they become incompetent and allow the blood to flow back into abnormal cardiac chambers (regurgitant lesion).

It is of importance to correlate, where possible, the pathological picture of endocarditis with valvular disease as encountered clinically. Thus, in acute endocarditis, the usual change consists in the production of ulcers with thickening of the cusps and in healed areas and granulations. Valvular deformity is slight or absent. In the longer-continued, subacute cases of endocarditis, verrucæ along the margins of the valves represent the prominent, pathological change. Valvular deformity is moderate. In chronic endocarditis, there is production of new tissue affecting not only the valves but also the *chordæ tendinæ;* the latter become stiff, the valves hard and inelastic. The result of both of these changes is marked deformity; in the

case of the mitral valves, there is lengthening and funnel-like formation of the mitral opening, accompanied by varying degrees of stenosis.

In a general way, it may be stated that the mitral valve is usually affected in simple, chronic endocarditis, and that the aortic valve is commonly involved in arteriosclerotic endocarditis and in syphilis (Chapter XVI).

**Myocardial Degenerations.**—The following are the chief pathological changes occurring in degenerative changes affecting the myocardium.

**Simple and Brown Atrophy.**—In both, the cardiac fibers become smaller and the entire heart is decreased in size. In addition, in brown atrophy, pigment is found in the form of small granules in the sarcoplasm. The entire heart is brownish in appearance. Both types occur in inanition and in various cachectic states. Brown atrophy is regarded as a senile change.

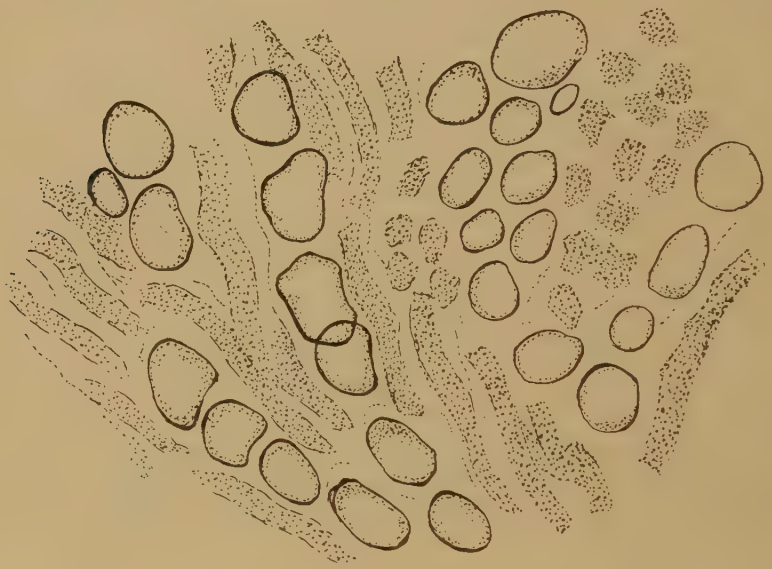

FIG. 28.—Fatty infiltration of heart muscle. (*After Kaufman.*)

**Parenchymatous or Albuminous Degeneration.**—Microscopically this condition is marked by cloudy swelling, with a varied number of albuminous, usually minute granules in the musculature. On gross section the cardiac musculature presents an opaque, dark red, somewhat spotted appearance, and is softer and more friable than the normal muscle. This degenerative change is a frequent accompaniment of infectious fevers, and of biological and chemical poisons. It is also found in severe anemia.

**Fatty Degeneration.**—This exists either as a primary process or it may represent a later development of parenchymatous degeneration. Depending upon its severity, the myocardium is studded with fat droplets of various size (Fig. 23). The musculature on section shows patchy or diffuse yellowish areas. When the damage is extreme, the muscle is flabby, friable

and grayish in color. Very many factors, especially those that alter the quantity and quality of the blood, may cause fatty degeneration. For example, it is found in severe primary anemias, in anemia following hemorrhage, in infectious diseases, in chemical intoxications (especially from phosphorous, arsenic and alcohol), in coronary disease, in cardiac hypertrophy and in chronic nephritis.

**Fatty Infiltration.**—The "Fatty Heart" (Fig. 28). This lesion must be distinguished from fatty degeneration. In the "fatty heart" the organ is covered by more or less extensive fat pads. Sometimes these lipomatous masses insinuate themselves between muscle bundles and appear as fat clumps or fatty spots under the endocardium. "Fatty hearts" are usually found in those obese, thick set individuals who have fat accumulations in other parts of the body. The subpericardial fat is sometimes considerably increased in quantity, so that the entire organ may be encased in the fat layer. The "fatty heart" is also occasionally found as a secondary change in the cardiac atrophy which accompanies cachexia.

**Coronary Lesions.**—Lesions of the coronary arteries are of supreme importance because of the prominent role played by disturbances of the intra-cardiac circulation in the etiology of myocarditis.

Infections such as typhoid fever, scarlatina and pyemia may so affect the media and intima of the coronaries that these coats undergo necrotic and hyperplastic changes, finally leading to patchy arterio-sclerosis of these vessels. Coronary endarteritis may give rise to extreme intimal thickening or calcareous deposits, with the deposition of thrombi on the denuded, roughened, intimal surface.

Whatever the underlying pathological process in the coronaries, the narrowed calibre of these vessels with consequent partial or complete coronary occlusion causes lessened arterial supply to the cardiac musculature. As a result, various degrees and kinds of myocardial changes may result. Such changes depend largely upon the amount of coronary occlusion, the rapidity of its formation and the infectious nature of the process; they may vary from anemia and myocardial degeneration to fibrous hyperplasia and even cardiac aneurism.

Coronary thrombosis occurs most frequently as an accompaniment of coronary arteritis and sclerosis. Occlusion and thrombosis of the coronaries at their orifices may also occur as the result of a neighboring aortitis, or from vegetations and clots on the aortic cusps.

Local anemia is the simplest primary change that results because of interference of the coronary circulation from emboli in the main coronaries or their subsidiary branches.

With complete obliteration of one of the smaller coronaries, the corresponding cardiac area becomes ischemic and pale, and undergoes coagulation necrosis, a process sometimes termed anemic necrosis or anemic infarct. The infarcted area may become myomalaceous, that is, the entire

affected musculature may become softened. In rare instances, such softened areas develop into aneurisms of the cardiac wall which may finally rupture. A more frequent outcome is organization of the anemic infarct; the necrotic material becomes absorbed, and granulation tissue with the production of new vessels takes place. This ends in fibrous or fibroplastic myocarditis, and finally, in scar tissue formation. If the process is widespread, the ventricular musculature appears striped on section. It presents a somewhat checkered appearance when older scars are found in conjunction with fresh necrotic areas.

**Myocardial Inflammation. Myocarditis.**—The primary inflammatory process involves the blood vessels and interstitial tissue, the musculature becomes secondarily affected by atrophy and degeneration.

**Acute Interstitial Suppurative Myocarditis.**—When occurring as a diffuse process from severe infections or from ulcerative endocarditis, there is widespread purulent infiltration of the cardiac musculature. Acute interstitial myocarditis is, however, more frequently a localized rather than a diffuse process, with single or multiple foci. The most common etiological factors are pneumonia, bacterial endocarditis and severe general infections.

As the result of bacterial infection in which a clump of bacteria forms the inflammatory nidus, an area of cloudy swelling, necrosis and fatty degeneration of the musculature is developed. If the leucocytes penetrate the necrotic zone, an abscess results. These abscesses are usually minute, though by confluence they may sometimes be readily seen with the naked eye. Small abscesses may occasionally end in scar tissue formation by destruction of bacteria, and by liquefaction and absorption of the pus cells. The defect is then covered by granulation tissue coming from the surrounding musculature. On the other hand, such abscesses may cause purulent pericarditis, emboli, ulcerative myocarditis and cardiac aneurisms. Any of these sequelæ can directly or indirectly cause death. In those instances in which the inflammation stops short of abscess formation, the process may end in the production of granulation tissue and of young connective tissue; finally scar tissue is formed.

**Chronic Fibrous Interstitial Myocarditis—Myocardial Fibrosis.**—This is a secondary productive process resulting in the formation of fibrous areas containing elastic and connective tissue. It largely originates as the consequence of disturbances of the intracardiac circulation from coronary disease. Other less frequent causes of this fibrotic change are chronic endocarditis and pericarditis, and bacterial and other infections. Experimental injections of bacterial toxins in animals have also been followed by the production of dense, fibrous connective tissue. Injection of adrenalin into the ear veins of rabbits has been followed by similar results. Depending upon the amount and type of coronary occlusion, coronary disease as already stated may give rise to degeneration or necrosis of the cardiac musculature with the production of fibrous tissue. This production of new connective tissue is therefore

not inflammatory in the more recent acceptation of the term, but is actually a (replacement) fibrous hyperplasia following atrophy of the muscle fibers; for atrophy is the primary effect on the heart of impaired circulation and nutrition either from coronary disease or from such others causes as decompensated valvular lesions. The new fibrous tissue may be present as small scattered foci or in larger, irregular patches (Fig. 29).

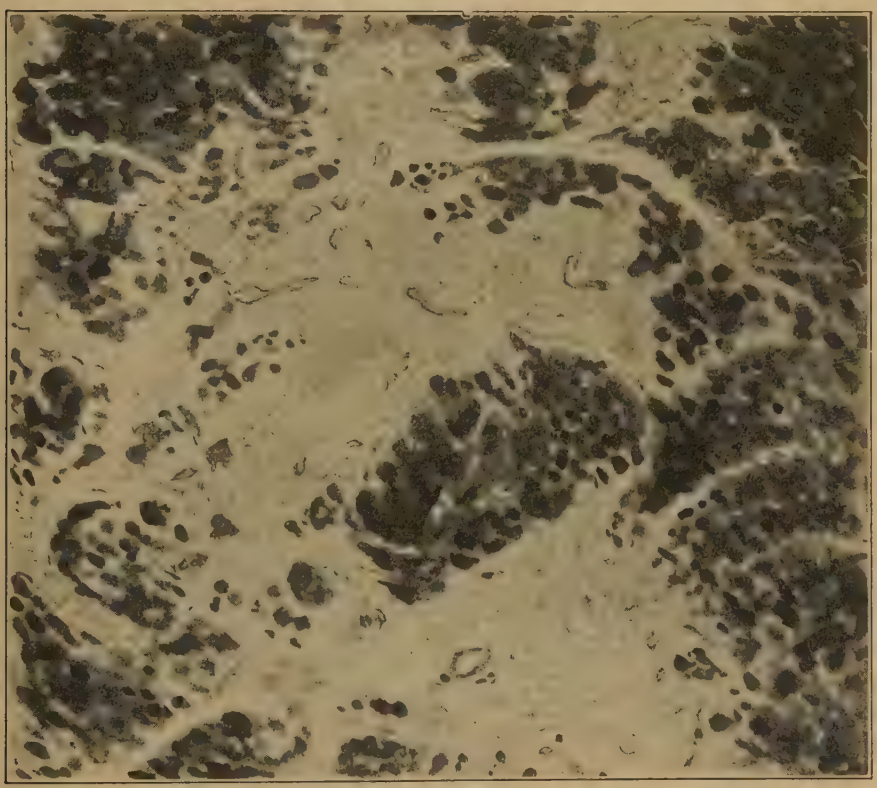

FIG. 29.—Chronic interstitial myocarditis. (*After Delafield and Prudden.*)

**Rheumatic Myocarditis.**—Interstitial fibrous myocarditis to a varying degree is also present as a concomitant of rheumatic endocarditis. Another type of myocardial change occasionally found in rheumatic endocarditis is the so-called Aschoff body or nodule (Fig. 30). The cells composing the nodules are epitheloid in character and contain large pale nuclei. Some of the cells resemble, and are of the size of plasma cells; others are much larger, resembling the giant cells of the lymph nodes in Hodgkin's disease. Intermingled with these cells there is occasionally a sprinkling of lymphocytic and eosinophilic leucocytes. The Aschoff bodies, when present, are particularly numerous in the interventricular septum and at the base of the left ventricular wall. Their presence in the former location is apt to involve the conduc-

tion system and therefore may cause heart block during the rheumatic attack. After the rheumatic attack has passed the Aschoff bodies change to fibrous tissue.

**Cardiosclerosis.**—Of late years there have appeared in the literature of clinical medicine the generic terms cardiosclerosis, cardiovascular disease, and cardiorenal disease. These are convenient although somewhat inexact terms which are meant to describe and group certain clinical conditions. There is no single pathological picture which accurately describes these groups.

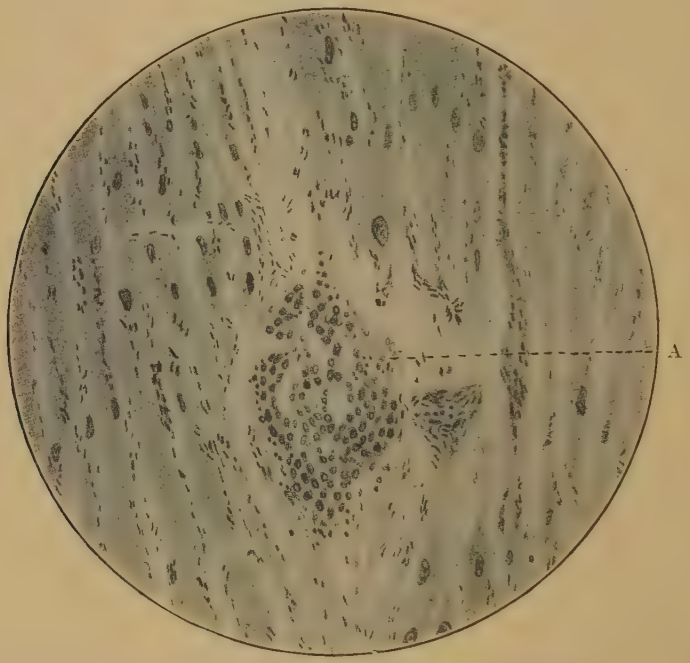


FIG. 30.—Nodules in the perivascular connective tissue in fresh rheumatic myocarditis. A. Aschoff nodula. (*After Aschoff.*)

As the names denote, the pathological change in the myocardium and endocardium is an exceedingly variable one. It depends to a great extent upon the amount and type of the other concomitant diseases affecting the kidneys and general arterial structure. The usual underlying factor is a general degenerative change of the entire vascular system, but especially of the smaller arterioles (capillary fibrosis of Jores) of the heart and kidneys. The heart then presents changes corresponding to this widespread cause, in addition to that wrought by renal disease. The heart is either atrophic, with a thickened, sclerosed and calcareous endocardium, and with indurated scars in the myocardium; or it is enlarged, the coronaries showing signs of mild or widespread disease varying from slight thickening to extreme calcification and obliteration of the arterial lumen. This arterio-sclerotic process

may involve the smallest coronary branches. The aorta is dilated, its intima thickened and atheromatous, containing calcareous deposits. A similar process affects the semilunar valves which, as a consequence, become incompetent (aortic regurgitation) or stenotic (aortic stenosis). The ventricular endocardium shows various patches of fibrotic thickening. Similarly when the process is widespread, the mitral valves are infiltrated with fibrous patches on their surface or along their free margins. The valves may even be incrusted with calcareous deposits. The myocardium presents indurated areas of scar tissue formation, or the entire musculature is riddled by massive formation of this character. Engrafted upon these commoner changes are those which frequently accompany hypertension and renal disease, *i.e.*, massive hypertrophy usually of the left but sometimes of both ventricles.

**Cardiac Syphilis. Spirochetal Infection.**—The immense strides in the detection of syphilis made possible by the discovery of the causative organism (spirocheta pallida) and by the use of the Wassermann reaction, have enhanced our knowledge of the frequency of lues as a cause of cardiac disease (Chapter XVI). The relation of syphilis to valvular disease, especially to disease of the aorta, although not proven, had long been suspected. Syphilis has now also been demonstrated as a fairly common cause of disease of the heart muscle itself; in fact, myocarditis is occasionally present in the secondary (Chapter XVI), as well as in the tertiary, stages of lues. By special staining methods, spirocheta have been found in the aorta and in the cardiac musculature. The most marked, and probably the earliest pathological involvement consists in a periarteritis of the arterioles of the coronary system, resulting in various types of muscle degeneration (fibrosis, fatty degeneration, brown atrophy). As a later involvement, gumma and gummatous infiltration also occur. The valves and walls of the aorta are, however, the site of predilection of cardiac syphilis. The picture varies from mild arteriosclerosis to extreme cicatrization with calcification. Thickening of the mitral cusps is less frequent; but it is occasionally sufficient to produce actual mitral regurgitation. Endocardial changes varying from slight opacities to extensive degeneration may coexist.

As a rare complication in the secondary stage of syphilis, it has been found, pathologically, that the myocardium alone may be occasionally involved. Such involvements are sometimes sufficient to produce symptoms of myocardial insufficiency:—dyspnœa on exertion, indefinite precordial distress and pain, precordial sensitiveness to pressure, slight pretibial edema, sometimes also a faint systolic murmur at the apex.

In occasional cases, isolated luetic valvulitis of the mitral or tricuspid has been found. In a vast majority of instances, however, the aortic valves, as stated, bear the brunt of the endocardial infection. In addition to the cusps, there is often widespread destruction of the aortic walls, finally resulting in aneurismal dilatations and in true aneurisms (Chapter XVI). Accompanying these are changes affecting the myocardium, the remainder of the endo-

cardium, and the coronaries. Together, they comprise a composite picture often similar to that found in non-luetic cardiosclerosis (Chapter XVII).

**Aneurismal Dilatations of the Aorta.**—The pathological process in dilatations of the first and second parts of the aorta consists of a mesaortitis, with perivascular infiltrations of the vasa-vasorum, small-celled or granulomatous infiltration in areas of the media, and splitting and destruction of the muscular and elastic tissue layers. Diffuse dilatation of the entire thoracic aorta from its root to the diaphragm is occasionally seen. I have observed three such cases: One was an autopsy finding and included severe myocarditis and coronary sclerosis, the other two were diagnosed by physical signs in conjunction with fluoroscopy.

While syphilis is the chief cause of aneurismal dilatations there are undoubted cases in which syphilis can be etiologically excluded by means of serological and clinical examinations (Chapter XVI). I have seen a number of patients—chiefly older individuals—in whom the Wassermann test was negative, who gave no history of rheumatism or syphilis, and in whom X-ray plates, fluoroscopic and clinical examination showed undoubted evidence of marked aneurismal aortic enlargement. These are probably examples of marked sclerotic changes found in the old in which the intimal coat is first affected; later the process may extend to the middle aortic coat (mesaortitis) and weaken it to ultimately produce aneurismal enlargement.

**True Aortic Aneurism.**—As in aneurismal dilatation, the chief pathological change is in the media. When the intima is also involved, it usually shows no atheromatous placques, but shallow, bluish tinted depressions, or the intima is pitted and puckered in small longitudinal or transverse ridges. There is perivascular infiltration of the vasa vasorum, and scattered leucocytic infiltration of the media. There is, further, destruction and separation of the elastic and muscular fibers; in other words a productive mesaortitis.

**Pathological Features of Streptococcus Viridans Infection.**—Of the chronic bacterial infections of the heart, that by the streptococcus viridans has been most exhaustively studied. The process is almost always engrafted upon a chronic rheumatic infection. Occasionally congenital lesions form the nidus. The pathological process consists of vegetative proliferative masses of grayish, greenish or pink color. Their main site is the mitral valves. Here they form a few soft, friable masses, or the valve may be encrusted with large polyoid lesions. The latter may then extend along the left auricular wall above, and the *chordæ tendinæ* below. The process on the *chordæ tendinæ* occasionally leads to ulceration and rupture of these structures. Similar sequelæ are sometimes found as the result of vegetations on the mitral. Proliferative lesions on the aortic cusps and walls are less common and less extensive than on the mitral. Mycotic aneurisms of the valves are also occasionally found.

Characteristic changes in the kidney depend chiefly on the presence of infarcts. When pyogenic, they give rise to numerous small congested areas

containing minute purulent foci; these are readily seen when the capsule is stripped from the kidney. Non-pyogenic bland infarcts occasionally occur; they show the changes usual to anemic necrosis; the infarcted areas are wedge-shaped and may be several centimeters in depth. When they are recent, the cut surface is yellow; the color becomes paler with the process of organization. Another type of infarct, sometimes assumed as pathognomonic of chronic streptococcus viridans, is embolic focal nephritis. Characteristic changes are then found in the glomeruli; a part of, or an entire tuft may be involved. The capillaries are congested, the glomeruli contain a fibrinous exudate, the glomerular epithelium becomes swollen and finally desquamates. The adjacent parietal layers of Bowman's capsule are often involved in the necrotic process, so that the entire necrotic area becomes semilunar in shape. The mass eventually organizes. There is a growth of epithelial cells from the healthy adjacent Bowman's capsule, finally covering the lateral surface of the mass. When healing is complete, the result is a hyaline area of pyramidal shape.

Cerebral lesions consist chiefly of areas of softening following emboli. Occasionally cerebral hemorrhages from rupture of embolic aneurisms of the cerebral vessels have been observed.

**Cardiac Hypertrophy—Etiological Considerations—Physiological Hypertrophy.**—True physiological hypertrophy of any muscle consists in an increase in the size of the normal cells. The process is probably intimately connected histologically with hyperplasia (increase simply in the number of normal cells) and with physiological hyperemia.

Based on our experience with skeletal muscles, we assume that the increased activity of the normal heart within certain limits will be accompanied by physiological hypertrophy of that organ. The assumption of physiological hypertrophy in man is also partly based on experimental observations, chiefly that in which the dog's heart was found considerably increased in weight as the result of working the dog steadily in a treadmill (Kulbs). The hypertrophy in these experiments was concentric, that is to say, about equally divided among the various chambers.

Increased cardiac activity can be aroused by various stimuli. The most obvious stimulus is one from the nervous system, for, as has been shown (Chapter III) there exists intimate correlation between nerve excitation and the various physiological cardiac functions. Another important stimulus to cardiac activity is a mechanical one, for simple stretching of the cardiac musculature by its contained blood incites the heart to contraction. Chemical changes produced by such drugs as digitalis and adrenalin, and chemical irritants assumed to be present in the blood of patients with exophthalmic goitre or renal disease may also act as excitants to cardiac contraction.

It is popular belief that physical work itself, such for example as is usual in athletics and laborers, can lead to physiological hypertrophy of a normal heart. Such assumption does not seem to take sufficient account of the

possible role of hypertension occurring during the actual performance of hard work as a causative factor of hypertrophy. Nor does it apparently emphasize sufficiently the possible incidence of infectious diseases (even old and long forgotten ones) as the nidus and primary cause of so-called "physiological" hypertrophy. In addition to the above considerations, most observations of "physiological" hypertrophy have been made upon the living, and unless controlled by modern methods it is now commonly conceded by students of cardiology that physical examination, even by the most expert clinicians, is open to gross errors and cannot vie with the much more exact roentgen and fluoroscopic examinations (Chapter XI). It is, for example, well known that percussion (Chapter XIII) as a criterion of the size of the heart is faulty, especially when we are dealing with slight or moderate hypertrophy in which it is assumed that there is no great increase in the size of the heart. Furthermore, all types of abnormalities such as accentuations, thrills and even diffuse apex beats—signs upon which the diagnosis of hypertrophy is commonly based—can be found in various forms of irritable heart (Chapter XVIII), in which that organ is of normal size. In this connection the following observation has an important bearing. A careful teleroentgenographic study of a series of soldiers' hearts who had undergone the exertions and physical strain incidental to warfare showed that the hearts were not larger than those of normal individuals (A. E. Cohn). Another observer, (Karson) who weighed the hearts of soldiers who died from septic infection, tentatively concluded that long-continued active service probably leads to an increase in the weight of the heart. It should be added however that arteriosclerosis, although mild, was present in nineteen out of the twenty-four hearts weighed; and the average weight of the enlarged hearts was 300 grammes, not much more than the normal.

Another cause of physiological hypertrophy emphasized especially by the German and French schools is the so-called hypertrophy of adolescent growth. It is claimed by these observers that in a certain number of such growing individuals, there is a physiological hypertrophic increase in the size of the heart. I have not been able to corroborate this assumption, for in many hearts of growing and large children, X-ray examination showed no inordinate or disproportionate enlargement as compared with the height and weight of the individuals. I would here except a few tall, young, rapidly growing boys from 13 to 16 years of age, of large bony frame, in whom I found by fluoroscopic examination that the heart was larger than the normal for such type of chest and for the size of these individuals. My impression of such enlarged hearts is, however, not that they are "hypertrophied" but that they are examples of organs which have grown larger and more quickly than the remainder of the skeletal musculature and hence stand out disproportionately. Whether these are to be classed as instances of "physiological hypertrophy" will of course depend on the conception of that term. These hearts in any case do not seem to become enlarged as a physiological reaction

to any assumed hyperemia or cardiac overstrain. In this connection, it is of interest to note that careful X-ray examination (the teleroentgenographic method (Chapter XII) by means of plates taken at a distance of seven feet) of a number of college oarsmen, some of whom had been rowing for several years, did not disclose enlargement of the heart beyond the normal for adults of that physique.

It is necessary to emphasize here that caution should be exercised in diagnosing cardiac enlargement by the usual physical signs. Such abnormal physical signs as an overacting apex (a condition often giving the impression of hypertrophy (Chapters XIII, XVIII) or over-vigorous cardiac activity are often due to unstable nerve equilibrium common to youth, a condition which may conceivably affect cardiac nerve control, and with it, cardiac activity.

Although based upon clinical observations which in their nature are not susceptible to proof, it seems probable to me that nerve exhaustion rather than physical exhaustion from expended physical energy is the controlling limit of physiological activity of the heart, and that physiological hypertrophy due to cardiac circulatory strain alone in a normal individual is probably a very rare occurrence.

**Cardiac Hypertrophy as the Result of Disease.**—According to Cohnheim's old classification, the types of hypertrophy were divided into the general concentric hypertrophy (equal distribution of the hypertrophic process to all the cardiac chambers), local concentric hypertrophy, and local excentric hypertrophy. This classification was based to a great extent upon the mechanical theories of overwork and of back pressure as the chief causes of ventricular hypertrophy. With the more modern conception of the etiology of cardiac hypertrophies, and because it has been found that the hypertrophic process by no means regularly follows the assumed overwork of a chamber consequent upon circulatory work and strain, the classification of Cohnheim has fallen into clinical disuse.

It has already been pointed out that varying degrees of myocarditis very frequently accompany valvular disease; this, however, does not necessarily imply hypertrophy.

Although subject to many exceptions, the following are types of hypertrophy to be found in the well-marked chronic cases of the respective valvular lesions. In mitral regurgitation, the prominent hypertrophy effects the left auricle; in mild cases, there is slight hypertrophy of the right ventricle, and slight or no hypertrophy of the left ventricle. In mitral stenosis, there is hypertrophy of the left auricle and of the right ventricle. In aortic insufficiency, tremendous hypertrophy of the left ventricle (*cor bovinum*) is the predominant enlargement, while in aortic stenosis, in the rare instances in which it is the only lesion, moderate left ventricular hypertrophy is the rule. Recent observations in which the ventricles were carefully separated by an exact method and were then weighed individually, have shown that

the normal weight relationship of right to left ventricle is as 1:1.7. By the same method of weighing, it was found that ventricular hypertrophy in mitral regurgitation was frequently general. In mitral stenosis it seemed that the back pressure theory had some justification, for in the pure uncomplicated stenotic cases, right ventricular hypertrophy was the rule.

Cardiac hypertrophy following chronic renal lesions with hypertension is an almost constant finding. The hypertrophy, however, is by no means constantly predominant in the left ventricle, as is commonly supposed, for the hypertrophy may be general; indeed, predominant right ventricular hypertrophy is by no means rare.

It is well to emphasize here that in valvular lesions the probable reason for frequent absence of the usually assumed predominant chamber hypertrophies is the accompanying myocardial disease, for the latter ultimately acts as the nidus for hypertrophy; that the myocardial (and hence hypertrophic) process may be scattered, or it may extend along and through the original complicated anatomy of the muscular cardiac layers (Chapter I) rather than follow the anatomy of the individual chambers; and finally, that back pressure, especially in mitral stenosis, less in aortic regurgitation, may play an important etiological part when the myocardium has first been attacked and affected by a diseased process.

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## CHAPTER V

## ETIOLOGY OF HEART DISEASE

**Classification.**—In the following enumeration, aortic disease will be included because histologically the intima of the aorta is a continuation of the endocardial endothelium; and disease affecting the one often affects the other.

Any classification adopted at this time must in a measure be arbitrary and tentative; for example, diseases now grouped as toxic alone may later be found to be of bacterial origin. Further studies may isolate specific factors in cases now grouped generically. With these limitations, the etiology of endocarditis and of cardiovascular disease may be conveniently tabulated as follows:*

1. Chemical agents
   - (*a*) Metallic and industrial poisons
   - (*b*) Alcohol
   - (*c*) Tobacco
   - (*d*) Diabetes
   - (*e*) Gout
   - (*f*) Products of food metabolism
2. Bacterial agents
   - (*a*) Diphtheria—pneumonia—typhoid—typhus
   - (*b*) Rheumatism—tonsillitis
   - (*c*) Pyorrhea—mouth infections—focal infections
   - (*d*) Pyogenic abscesses.
3. Bacterial endocarditis
4. Spirochetal infection—cardiovascular syphilis
5. Endocrine disturbances
6. Senility

### 1. CHEMICAL AGENTS

(*a*) **Metallic Poisons.**—Of metallic poisons, the action of lead is best known. Lead poisoning (plumbism) formerly occurred principally in painters who worked with pigment containing much lead. In modern pigments, however, the amount of lead has been reduced to a minimum. Die workers and those working in fumes in which lead is used now form the chief susceptible class. Lead poisoning may affect the endocardium and aorta. In rare instances, the coronaries and their branches also become diseased. The renal arterioles are often involved, and nephritis results. Depending on the severity and location of the arteriolar disease, the symptoms and physical signs may be those of aortitis, nephritis, cerebral endarteritis, coronary disease or of endocarditis; or there may be a clinical picture com-

* The clinical phases of these various poisons and factors of heart disease are described in their appropriate chapters. (See Table of Contents.)

bining these pathological entities in varying proportions. The patient's occupation, a history of colic, peripheral neuritis, the presence of a lead line on the gums, microscopic examination of the blood, and the chemical examination of the feces and of the total urine of several successive days for lead are data which are of aid in the search for lead as the etiological factor. These examinations, however, are of use chiefly during the time the individual is still working at his lead trade; for it must be remembered that cardio-vascular disease originating in lead poisoning may insidiously progress long after the patient has given up his original occupation. Whether myocardial degeneration, when present, is primary, or is secondary to disease of the coronary system is still an unsettled question. However, the selective action of lead on the arterial system seems to indicate that myocarditis results secondarily from the vascular disease.

Severe phosphorous poisoning can produce fatty degeneration of the heart and of the arterial intima. Other metallic poisons—copper, mercury, arsenic—have a much greater selective action upon the gastro-intestinal canal, and upon nervous, osseous, and renal, rather than upon the cardiac structures. Except for the action of mercury upon the kidneys, it is questionable whether copper, mercury and arsenic produce cardiac damage that is clinically recognizable.

**"Industrial" Poisons.**—I am applying this term to possible other poisons, non-metallic in nature, which may have some as yet practically unknown effect upon the cardio-vascular system. Thus the fumes of some aniline dyes, some of which in acute poisoning cause marked blood and other destruction may also insiduously change the chemical composition of the blood, and finally cause disease of the myocardium by interference with its proper nutrition. These and other similar questions are still problems of the future in the study of the possible effect of industrial poisons upon the heart.

(*b*) **Alcohol.**—The degree of damage to the cardiovascular system by alcohol is still a matter of dispute. Like other poisons, it affects the cardiovascular structures to a variable extent, so that necropsy reports showing absence of cardiovascular damage in chronic alcoholism are not necessarily evidence of its innocuousness. It is probably true, however, that the importance and frequency of alcohol as a cardiac poison have been overestimated. Alcohol attacks particularly the cardiac musculature; the result is myocardial disease varying from slight fatty degeneration to scar-tissue formation. The arterial system, when affected, presents various grades of thickening of the intima as well as calcareous deposits; in exceptional cases, there is also degeneration of the remaining arterial coats.

The clinical symptoms referable to alcoholism are usually those of myocarditis and myocardial insufficiency. The heart is moderately hypertrophied. As a clinical syndrome of moderate myocarditis may be cited that due to constant drinking of large amounts of beer (the "Munchener Bierherz"). In severe cases of alcoholic myocarditis, cardiac hypertrophy may

be extreme. In one case that I observed at necropsy, a man of 45 who was an inveterate wine drinker, the ventricular musculature consisted mainly of scar tissue, the heart weighed twice the normal. Symptoms and clinical signs due to aortitis—an impure first, and an accentuated second sound at the right base, and hypertension—are also occasionally due to alcoholism.

(*c*) **Tobacco Poisoning.**—It is the general belief that tobacco users are particularly prone to aortic disease, coronary sclerosis and myocardial degeneration, and that these pathological changes are caused by nicotine, the main tobacco alkaloid. This view is based chiefly on the result of experiments on rabbits in whom nicotine solutions were injected; as a result, various degrees of aortitis were produced. Before conclusions from such experiments can be regarded as comparable to tobacco poisoning in the human being, it should be remarked that these injections were repeated at frequent intervals over a long period before aortitis resulted. As further objections to inferences drawn from these nicotine injection experiments, it must be stated that others have found no abnormal aortic change following nicotine injections; indeed, one observer found spontaneous changes in the aorta in a certain proportion of normal rabbits. Moreover, when the attempt was made to produce parallel conditions by forcing rabbits to inhale fumes on successive days from measured amounts of burning tobacco, no definite changes in the aorta were found. One observer who made a careful pathological study of the hearts of heavy smokers dying of various diseases found only a slight change in the papillary muscle; this he attributed to the rapid heart action usually found in smokers. From the experimental and pathological sides, therefore, it must be concluded that no incontrovertible proof has as yet been adduced that smoking in itself produces cardiovascular disease. On the other hand, as the result of physiological and pharmacological experiments, it is known that nicotine has a strong affinity for nervous structures (neurotropic action), particularly upon the sympathetic nervous system (Chapter X). Many of the symptoms complained of by patients with so-called "tobacco heart" (Chapter XXVI)—tachycardia, arrhythmias of various types (especially extrasystoles), syncope, precordial pains and distress—can be more readily and rationally explained, I believe, by the selective action of nicotine upon the sympathetic nerves and ganglia (Fig. 210) than upon the assumption of organic cardiovascular disease. In the majority of cases, when tobacco is withdrawn, cardiac irregularities and pain cease, and patients examined months or years later show no evidence of organic damage to the heart or arteries. I believe, however, that if from any other cause a symptomless aortitis exists, the lesion may become activated and produce symptoms as the result of tabagism. Even this statement is only surmise, for it is not susceptible of clinical proof.

(*d*) **Diabetes** is frequently accompanied by arteriosclerosis and nephritis. In chronic cases the myocardium may show advanced fatty degeneration. The rather marked tendency to arteriosclerosis—and therefore to coronary

disease—may be a primary cause of myocardial change found in some diabeties. The clinical symptoms and signs of the resultant cardiovascular disease, when not marked or complicated by diabetes itself, are high blood pressure; thickening of the palpable arteries; sugar, albumen and casts in the urine, and aortitis. Therapy should be directed to both diseases—diabetes and cardiosclerosis; relief or cure of the former is often followed by beneficial results in the latter.

(*e*) **Gout** is very frequently accompanied by arteriosclerosis, nephritis and hypertension. Aortitis and coronary disease are also very common in gouty individuals, and as a consequence, myocardial changes of various degrees and extent are often present. An abnormal amount of uric acid in the blood and in the tissues may be the cause of these pathological changes, or both the latter and gout may be the expression of some common, as yet unknown, underlying factor or diathesis. The problem of therapy directed to the cardiorenal disease is rarely complicated by the presence of gout.

(*f*) **Products of Food Metabolism, Amino Acids, etc.**—The study of metabolism and of blood chemistry has shown the presence of amino-acids and other protein derivatives in the blood. Experiments have demonstrated that protein feeding can produce changes in the cardiovascular and renal apparatus of animals. Although clinical proof is lacking, these investigations suggest that protein overfeeding in man may be followed by arterial disease, and that therefore protein can act as cardiovascular poisons.

### 2. BACTERIAL AGENTS

In the production of disease, bacteria act directly or by the elaboration of chemical poisons, that is, of toxins. Chemically the latter are at present regarded as extremely complex poisons of nitrogenous nature. It should be emphasized that the various bacterial agents possess special predeliction for various parts of the cardiac structures and hence cause disease of varying degrees (*e.g.*, of heart muscle, endocardium, conduction system, etc.).

(*a*) **Diphtheria.**—The heart muscle in severe diphtheritic myocarditis undergoes fatty and parenchymatous degeneration; the valves and endocardium are rarely affected. When the poison attacks the heart more slowly, there is in addition, leucocytic infiltration (inflammatory myocarditis). Occasionally the conduction system is attacked, with the consequent production of various types of heart block (Chapters X, XI). The clinical syndrome of the heart in diphtheria is described in another connection (Chapter XXVI).

**Pneumonia.**—Endocarditis due directly to an invasion by pneumococci is by no means rare; this type will be discussed later. The pathological damage is comparable to that found in bacterial endocarditis (Chapter IV). Regarding the effect of pneumonic toxins on the heart, many pathological examinations of the cardiac musculature have been reported. Slight paren-

chymatous degeneration (cloudy swelling) is the rule; occasionally more severe damage has been described. When widespread, these degenerative changes can undoubtedly cause death. But there is no evidence that slight cardiac degeneration, the usual damage in pneumonia, is in itself sufficient to produce death; nor have attempts been made to correlate the amount and degree of pathological damage with the clinical or bacteriological evidence of toxicity. Experimental evidence pertaining to this subject indicates that pneumonia usually acts as a poison upon the functional power of the heart; for example, pneumonic blood perfused into a healthy dog's heart is immediately followed by greatly weakened contractility. The contractions become normal when healthy blood is subsequently perfused. At present the conclusion seems warranted that death from pneumonia in the majority of cases is not due to demonstrable changes in the myocardium. On the other hand, damage to the cardiovascular apparatus as a *late* sequel of pneumonic infections has been insufficiently emphasized. Such sequalæ produce symptoms only some months or years after pneumonia has run its course, so that the connection between the two diseases is usually entirely overlooked. In some instances the pneumonic poison seems to light up a dormant cardiovascular process; in others, it is the prime factor. Case reports illustrating these statements are given elsewhere (Cardio-vascular Clinics).

The action of diphtheria and of pneumonia as cardiovascular poisons has been described in some detail because similar observations apply in varying degrees to other infectious diseases. Typhoid fever deserves special mention in this connection, for it occasionally causes areas of necrosis in the musculature.

All these various toxic causes of myocarditis show the importance of a careful search for infectious disease as an etiological factor, especially when the etiology of cardiac impairment is obscure.

(*b*) **Rheumatism—Tonsillitis.**—Of all diseases, rheumatism is the most frequent source of valvular disease. Although the etiology of rheumatism is not yet known, recent experimental research and clinical observation indicate that it is of bacterial (probably of streptococcic) origin. For the present, however, until the specific organism has been isolated, it is here classified under toxemias. Valvular endocarditis is the most frequent sequel of a rheumatic infection. Varying degrees of myocarditis, and, occasionally, the formation of submiliary inflammatory nodes (Aschoff's bodies) are frequent accompaniments of rheumatic endocarditis. Permanent damage always results from rheumatic valvulitis and myocarditis; but here, as elsewhere in the body, if the infective process stops early, scar tissue may form (Chapter IV); hence clinical signs of myocardial or valvular disease may never appear. Necropsy alone gives evidence of cardiac damage in such instances. In some cases, even if clinical manifestations of valvular disease, particularly of mitral regurgitation be present, the process may heal, the

valvular leakage may stop and the patient remain clinically well. Such observations at the bedside have been substantiated by necropsy.

Tonsillitis is grouped with rheumatism in the etiology of cardiac disease, for, like rheumatism, it may also give rise to joint manifestations and to endocarditis. Even without joint involvement, tonsillitis frequently causes endocarditis. In fact, exceedingly mild and apparently harmless tonsillar and pharyngeal attacks, accompanied by a few spots on the tonsils or pharynx, can be the source of an endocardial infection. As one instance, a vigorous young man of twenty-one developed mild pharyngeal grippe lasting three days; the highest temperature was 101.5°. Within one week typical signs of a mitral regurgitant lesion were present. Similarly so-called "colds" (acute pharyngeal and nasal catarrh)—can occasionally, although rarely, be the infectious nidus for endocarditis. (See Cardio-vascular Clinics.)

(*c*) **Pyorrhœa Alveolaris—Mouth Infections—Focal Infections.**—Much experimental and clinical work has recently been done to show that the various pyorrhœa-producing organisms harbored in the mouth may, by systemic absorption, cause myocarditis and endocarditis. Among other bacteria, the streptococcus viridans has been especially accused. However, it has been shown that this organism is a fairly frequent inhabitant of the *normal* mouth; hence not only demonstration of the pyogenic organism is required but also definite clinical evidence that it bears an etiological relationship to the cardiac disease. In the face of negative blood cultures and in view of the other well-known causes for endocarditis, several procedures are necessary in order to prove the correlation between pyorrhœa alveolaris and cardiac disease. Thorough examination of the mouth for carious teeth and for periostitis by radiographic examination of the jaws and of the roots of the teeth constitutes the first step. Then a careful search of the entire body must be made for other possible infective foci which may cause cardiovascular disease. Cholecystitis, cystitis, deep-seated bone abscess and pyelitis are some of these inflammatory conditions. So that even should roentgenography disclose root abscesses, and the mouth harbor pathogenic bacteria, it is for the clinician to decide from the clinical history, the type and probable duration of the cardiovascular disease, and from a complete examination for all other possible sources of infection, what weight, etiologically, should be given to the presence of root abscesses or pyorrhea as the causative agents of endocarditis. Thus studied, it appears to me that the claims now made for the extremely frequent connection between oral infections and heart disease are unwarranted. This statement indeed fits in with general clinical experience. For instance, very many children with endocarditis have healthy teeth and mouths, and again many with carious teeth have normal hearts. In those patients with cardiovascular disease observed by me, in whom extraction of teeth had been done because of root abscesses, extraction had no effect upon the cardiovascular process or upon the clinical signs. In general, one would not expect that the small amount of toxins presumably elaborated

and absorbed from a small dental focus could be a frequent cause of endocarditis; one would expect sufficient antibodies to be developed to prevent cardiac mischief in the great majority of cases. On the other hand, it cannot be denied that, given an extremely susceptible individual, general systemic damage and especially cardiac disease can occasionally, though I believe very rarely, be caused by diseased teeth or diseased gums acting as foci of infection. It is therefore necessary for the clinician to weigh all factors and not to conclude too hastily that because pus, pathological bacteria, or small root abscesses are found in the mouth, they are necessarily the cause of the cardiovascular disease in that individual. We are greatly indebted to the dental profession for indicating the teeth as a "focus" for endocarditis, but the physician must finally judge all the evidence in every case before he can decide whether the cause of endocarditis lies in the mouth or elsewhere. None of the statements here made of course militate against the fact that purulent mouth conditions should receive proper dental attention in order to remove that source of a possible endocarditis. My main object has been to refute the usual accepted opinion that diseased teeth *commonly* cause disease of the heart. In those exceptional cases under my observation in which alveolar pyorrhea might have been the cause of the existent cardiovascular disease, the pyorrhea was frank and usually severe even at the time the patients presented themselves for examination because of cardiac symptoms. There was usually a long history of foul breath, and of decaying and loose teeth. The patients were of middle age or past middle age. Questioning elicited that the pyorrhea was severe and of several years duration. Of interest and importance is the fact that, in my own observation, at least symptomatically and pathologically, the picture is one of cardiosclerosis (Chapter XVII). It seems as if the poison acts, if at all, primarily upon the arterioles of the cardiovascular system, affecting in varying degrees the vessels of the heart and kidneys. Its final effect upon these organs often closely resembles, indeed sometimes coincides with, the pathological picture of senile cardiosclerosis (Chapter XVII).

Regarding the relation between diseased teeth and valvular endocarditis in children, with the exception of cases of widespread and gross oral sepsis developing into bacterial invasions of the blood, there is as yet no definite evidence that diseased teeth have actually caused endocarditis. Such a possibility cannot of course be denied, particularly perhaps in valves already damaged by rheumatism or other infections. The warning lies in accepting possibilities for probabilities, and in assuming that because diseased teeth occasionally produce rheumatism of a certain type, they therefore can act as commonly and as virulently upon the endocardium in the production of valvular disease. Each case must be carefully studied individually, and although thorough modern dental care should be given abscessed teeth, we should not lean to unnecessary wholesale extraction in our efforts to rid the mouth of "foci of infection."

The clinical aspects of "foci of infection" are described in other connections. (See Table of Contents.)

(*d*) **Pyogenic Abscesses.**—These may occasionally produce endocarditis by the production of a bacteremia or from toxins. In endocarditis where a frank etiological factor cannot be found, careful search should be made for hidden abscesses as possible foci of infection.

### 3. BACTERIAL ENDOCARDITIS

Bacteria were demonstrated in valvular vegetations by Heilberg as early as 1869. Since then many type of organisms have been recovered from such vegetations, and more recently, isolated from the blood.

The question of systemic bacterial infection in rheumatic fever is still unsettled, although, as already indicated, there are good clinical and experimental grounds for considering it a bacterial disease.

The known organisms found in the blood which produce endocarditis are grouped, according to Simon's modification of Litten's classification, as follows:

1. Ordinary streptococcus.
2. Small streptococcus. (Probably all alike, but variously named by different observers.)
   - Streptococcus viridans (Schottmueller).
   - Endocarditic cocci (Libman).
   - Modified pneumococci (Rosenow).
   - Saprophytic streptococci (Horder).
   - Streptococcus tenuans (Hastings).
3. Staphylococcus albus and aureus.
4. Pneumococcus.
5. Gonococcus.
6. Meningococcus.
7. Bac. coli.
8. Bac. influenzæ.
9. Bac. pyocyaneus.

As gathered from the statistics of various authors, the most common invaders in *acute* bacterial infections are, in order of their frequency, the streptococcus pyogenes and the staphylococcus aureus; next in frequency are the pneumococcus and gonococcus; the streptococcus viridans is very rare. The other bacteria—staphylococci, meningococci, bac. coli., bac. pyocyaneus, and bac. aerog. capsulat.—are only occasionally found. The *chronic* invaders are, in the order of frequency, the streptococcus viridans (common), the streptococcus pyogenes, pneumococcus, bac. influenzæ, and the gonococcus. It is thus seen that the gram-positive chain cocci are the chief causes of bacterial endocarditis.

Regarding the relative frequency of bacterial invasions of the various valves, the mitral is the most frequently affected alone; next is the aorta. Bacterial infection of the pulmonary or tricuspid valves alone is exceedingly rare. The most common combination of a multiple infection is the mitral

and aortic; the next, the tricuspid and mitral. Vegetations on the walls of the auricle and ventricle have also been described, but they do not occur without vegetations on the valves as well.

### 4. SPIROCHETAL INFECTION

As already noted, and as is well known, the etiological factor is the spirocheta pallida. The pathological changes in the heart produced by this organism have already been described (Chapter IV).

### 5. ENDOCRINE DISTURBANCES

Because of our incomplete knowledge of the endocrines, there are at present no exact data of the effect of disturbances of the ductless glands upon the etiology of cardiac disease. An exception is the thyroid gland. Here there is definite evidence that in addition to the symptoms caused by hyperthyroidism (Chapter XVIII) there may be not only functional myocardial disturbances but occasionally actual myocarditis may be present. There is another fairly frequent distinctive picture of cardio-vascular disease; that found in women at the menopause. Such patients may have hypertension for years (Chapter XXV); the disturbance seems mainly vaso-motor in origin. The question arises whether such types of hypertension—often innocuous in the beginning—may finally produce actual cardio-renal disease. Until autopsy reports of such conditions have been offered, the question must remain *sub judice*.

### 6. SENILITY

It is commonly assumed that old age as such forms part and parcel of the chronic diseases and changes of the heart and arteries so frequent in the old. The general trend of this thought, so far as it can be translated, is that the "wear and tear of life"—especially from the physical standpoint—has finally caused "senile" cardiac changes. This view seems incomplete and inexact; in particular, it does not reckon sufficiently with factors of infection, perhaps present many years before and long since forgotten, which can ultimately bring about definite pathological changes in old age. This phase of the subject will probably not be cleared up until we can obtain experimental or clinical evidence of the slow, insidious progress of the *effects* of infection long after the infection itself has completed its acute course. Perhaps then senility as far as the arterial tree is concerned may be translated into the insidious results of the long-continued "wear and tear of life" acting upon arteries already affected, no matter how minutely, by some actual pathological change, a minor effect of any one of the commoner, universal infections.

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## CHAPTER VI

## DYNAMIC AND MECHANICAL CONSIDERATIONS—CARDIAC DILATATION

**Valve Closure.**—The valves guarding the mitral and tricuspid orifices are slightly muscular at their origin but are otherwise membraneous (Chapter I). They are attached by means of the *chordæ tendinæ* to the apices of the papillary muscles (Figs. 11, 12). The tendinous cords and papillary muscles serve to keep the valves under a certain amount of tension so that they cannot become inverted into the auricles during any phase of the cardiac cycle. During cardiac systole, the *chordæ tendinæ* become taut, chiefly as the result of contraction and shortening of the papillary muscles. The semilunar valves guarding the pulmonary and aortic orifices form a complete membraneous closure at these openings, the small fibrous nodules at the center of each cusp (*the corpora Arantii*, Fig. 12) helping to make the closure perfect. The valves of the great vessels and of the ventricles very probably do not flap back during diastole sufficiently to touch the arterial or ventricular walls, respectively. Such approximation is presumably prevented by eddies occurring during diastole which tend to float the valve leaflets away.

The usually accepted view regarding the mechanism of valve closure is that with the inrush of blood, these diastolic eddies not only float up the valves to prevent their touching cardiac structures but in addition they co-apt the various valve leaflets to bring about valve closure. A more modern conception assumes that accompanying the diastolic inrush of blood immediately after systole, negative pressure is momentarily induced; it is this negative pressure which tends to draw in blood behind the valve leaflets; as a consequence the valves close. It seems probable that this action permits of perfect closure because it begins at the attached border of the valve leaflets and thus tends to hermetically seal the various valve openings.

**Pressure in the Cardiac Chambers.**—Various instruments—optic manometers, cardiometers, etc.—have been devised for the study of pressure curves as they exist in the chambers of the animal's beating heart. As a result of these studies, it has been determined that the pressure in both ventricles is practically the same. The initial rise in intraventricular pressure (Fig. 57) is initiated by a very slight rise in presystole due to auricular contraction. After this there occurs a sharp rise in intraventricular pressure. This takes place during what is called the isometric period of the cardiac cycle—that brief interval during which both the auriculo-ventricular and semilunar valves are closed, and the cardiac fibers are in a state of tension before actual ventricu-

lar systole begins. During ventricular systole and with the opening of the semilunar valves, the intraventricular pressure curve reaches its peak. With the onset of ventricular diastole, the intraventricular pressure rapidly falls, to again reach its base line with the onset of auricular systole.

There is a sharp rise of pressure in the aorta and pulmonary artery as the blood is ejected with the onset of ventricular systole (Fig. 57); this pressure is sustained during the entire period of ventricular contraction. With the onset of ventricular relaxation and diastole, the arterial pressure rapidly falls. As in the ventricles, so also in the auricles there is no appreciable difference in the pressure curves of the right and left sides. There is a sharp rise of auricular pressure due to auricular systole, followed by a fall caused by auricular relaxation and diastole. With the onset of, and during ventricular systole (at which time the auricles are filling with blood), there is a steady slight rise of intra-auricular pressure as the result of stasis.

**Volumetric Ventricular Curves.**—For the experimental observation of volumetric curves in various phases of the cardiac cycle, a cardiometer is employed. This is a glass vessel containing a rubber dam or diaphragm which can be snugly closed around the auriculo-ventricular groove. The projecting end of the cardiometer is placed in circuit with a tabular recorder and revolving drum. Thus a graphic study may be made of the systolic output as well as of the volumetric changes occurring during diastole. With the ejection of blood from the ventricular chambers during systole, there is naturally a great diminution in their size. The period of ventricular emptying and filling may be divided as follows: (1) the stage of blood ejection: the systolic period. This is followed by (2) the early diastolic filling, called also the active diastole or diastasis (Henderson); during this period, filling proceeds rapidly; finally there is (3) the late diastolic filling period (passive diastole). As shown by experiments, the entire diastolic period (both active and passive) is profoundly influenced by variations in venous pressure and cardiac tone, and by the cardiac rate, that is, by the number of cardiac cycles per minute. These three factors are by no means interdependent although each by itself can markedly affect the ventricular volumetric curve. Regarding the influence of rate, the maximal systolic output is seen when the heart beats at the rate of 75 or 80 per minute. At higher speeds, especially with extreme cardiac acceleration, the diastolic period (especially active diastole) becomes considerably shortened, and the systolic output markedly reduced. The output per minute may still increase but not proportionately to cardiac acceleration.

Regarding the influence of venous pressure on the volumetric curve, no "critical" pressure has been discovered which will always deleteriously effect the volumetric curve. However, an amount of venous pressure can be induced during which the cardiac output no longer maintains its level, but decreases, and with it, the heart dilates. Muscular exercise (that is, the action of skeletal muscles as an accessory pump in forcing the blood through

the systemic veins) exerts a great influence in filling up the right auricle, thus enabling the right ventricle to receive its proper quota of blood.

Finally, regarding the influence of cardiac tonus on the volumetric curve, it must be remembered that tonus (Chapter III)—that state of partial contraction in which variations in size of the heart are resisted—is difficult to determine experimentally. It seems probable, however, that cardiac tonus must exert a decided influence on systolic output and volumetric filling. Thus, with high tonus, the systolic output is quite effective but diastolic filling is interfered with, while with low tonus, there is rapid diastolic relaxation and filling.

**Acute Cardiac Dilatation.**—The importance of the physiological and experimental considerations just described lies in the attempt to correlate them with what actually occurs in the human heart in health and disease.

Some facts regarding the effect of exercise upon the size of the heart are pertinent in this connection. Previous attempts at exact studies of the heart by means of X-rays have not given any conclusive evidence as to the size of heart during actual exercise or work, partly because pictures were not taken during the exercise and partly because the phase of respiration during which the roentgenogram was taken was not considered. A roentgenogram taken, for example, with the diaphragm in the act of rising will naturally push up the heart and show a much broader cardiac shadow than if the heart picture is taken with a descending diaphragm (Chapter XII). Exact teleroentgenographic study of hearts immediately before, and about 20 seconds after exercise with the respiratory phase taken into consideration, showed slight diminution in the size of the heart in 29 out of 33 normal subjects. Attacking the problem of cardiac output from the physiological standpoint by a study of the tension of nitrous oxide experimentally inhaled, and measurement of the quantity exhaled, it has been demonstrated that the cardiac output in the resting state is 3 to 5 liters per minute and that during heavy muscular work, the cardiac output per minute may reach 20 liters. This of itself shows the tremendous "factor of safety" of the human heart. Other physiological observations have demonstrated that there exists a normal physiological dilatation of the heart during exercise, and that its limit is fixed by that amount which fills the pericardial sac during diastole (Bainbridge).

Naturally it is a very difficult process to determine actual dilatation clinically with any exactitude, so that we are often limited to inferences alone. Indeed one observer (Williamson) who made very careful teleroentgenographic studies of both normal and decompensated hearts during exercise, categorically states that even by this refined, objective method the amount of dilatation of decompensated hearts after exercise is so small (3 mm.) that it is obviously all but impossible to detect it objectively at the bedside. The cardiac fibers possess the property of shortening during, and of lengthening after, contraction. In health this function is perfect. Fundamentally, it is this ability to contract and expand which indicates what is known as the

"cardiac reserve." As the result of valvular disease, there is a varying amount of change in the ventricles and their chambers; this consists of myocarditis, hypertrophy and dilatation. These abnormal factors compromise to a varying degree the underlying function of lengthening and shortening of the cardiac fibers, and hence interfere with the reserve power of the heart. In consequence, the cardiac power is decreased and signs of decompensation (Chapter XIV) appear. There is also evidence that differences in tone may play a part in the symptomatology. For example, patients may complain of dyspnœa, of vaso-motor symptoms, and even of precordial distress without any physical or clinical evidence of cardiac dilatation. These symptoms may appear suddenly, remain for a few hours or a day, and then disappear without any signs of decompensation.

Several pertinent questions arise. To what extent in the living do such experimental factors as venous pressure and cardiac rate increase or lessen output? If output is lessened, does it cause the gradual or rapid onset of cardiac dilatation? What is the role of that much misused term, tonus, in cardiac dilatation? The matter is further complicated by the fact that it is by no means easy to diagnose cardiac dilatation by the usual, or indeed by refined methods of examination. In fact, I believe that cardiac dilatation in the sense of mechanical stretching of the heart is often assumed when its actual presence is, to say the least, purely inferential. An exception to this statement, however, must be made with paroxysmal tachycardia (Chapter X). In some of these patients the heart actually swells, so to speak, so that the presence of dilatation can usually be diagnosed by the ordinary methods of examination. The heart occupies an abnormally large area in some individuals during the tachycardial seizure, and recedes in size when the paroxysm ceases.

A further perplexing clinical aspect of the problem of cardiac dilatation is the fact that we possess no data as to the amount of acute dilatation a diseased heart can stand, for cardiac disease in itself can presumably decrease heart tone and thus establish a vicious circle. Again, a heart already inured in a way to rapid heart action, may be able to withstand more dilatation without serious results than one in which the tachycardia is sudden and unexpected. I believe that the subject of cardiac dilatation in its clinical sense—especially its diagnosis and degree—presents very many perplexing phases, each of which has to be carefully weighed, and indeed often only inferred in order to arrive at the proper diagnosis in the case at hand.

Cardiac dilatation and cardiac reserve power are also intimately connected with the involved question of so-called cardiac strain. The subject will be dealt with more fully in other connections (Chapter XIV). Here it is sufficient to indicate that in normal hearts, the neurogenic nerve control and cardiac inhibitory centers are the safeguards and checks that probably prevent abnormal cardiac dilatation. That is to say, before the cardiac

chambers became greatly overdistended, a degree of nerve exhaustion supervenes which prevents further overaction, tachycardia, etc.

To sum up the dynamics of a normal cycle and its clinical application to cardiac dilatation: The presssure in the ventricles rises with ventricular systole and falls with the beginning of ventricular relaxation (diastasis). When the auricular exceeds the ventricular pressure, the auriculo-ventricular valves open and blood flows from auricle to ventricle. The rate of ventricular filling depends on the degree of ventricular relaxation and on the venous "head" (venous pressure). As the pressure of the auricle becomes increased by the venous flow and tends to equal that of the ventricle, the flow becomes less rapid until auricular diastole is finally completed and the auriculo-ventricular valves close. Cardiac dilatation unless extreme is not readily diagnosticated. Among important factors in its causation are the cardiac rate, venous pressure and tonus. In a majority of instances, it is impossible to weigh these factors categorically. Often a shrewd analysis is the only clinical solution of the problem. Until we possess more knowledge and are able to diagnose slight and moderate dilatations, and their effects on normal and diseased hearts, the term cardiac dilatation should be used cautiously.

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## CHAPTER VII

## GRAPHIC METHODS—SPHYGMOGRAPHS AND SPHYGMOGRAMS —THE INK POLYGRAPH—POLYGRAPHIC CURVES—RADIAL CURVES.

For purposes of exact graphic study of the time relationships of arterial and venous pulsations, various instruments have been devised. Among the first to construct and employ an instrument for the registration of the flow of blood through the heart and arteries was Marey. He, together with his colloborator, Chauveau, constructed special sounds armed with rubber capsules at one end; these were introduced into the arteries at the base of the heart and into the heart cavities. The other ends of the sounds were con-

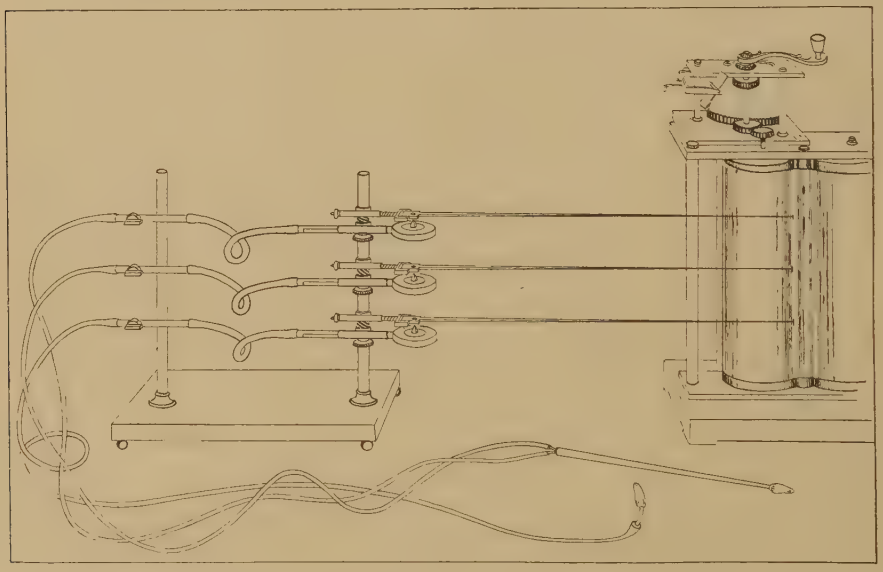

FIG. 31.—Apparatus for experimental curves in animals and for registering pressure curves as well as the time relationship of the events in the cardiac cycle. Sounds were placed in various cardiac chambers; the movements of the lever were registered upon the paper covering the revolving drum. (*After Marey, one of the early experimental investigators.*)

nected with rubber tubing which was connected with tambours and writing levers. The levers rested against a revolving drum covered with blackened paper (Fig. 31). In this manner, movements imparted to the capsule were transmitted to the lever and were thus transcribed on the revolving paper.

In the various experiments undertaken upon the horse, the sounds were placed into the right auricle, or into the right and left ventricles. Thus curves were obtained that were measures of the pressure as it actually existed in the cardiac chambers; in addition, the time relationships of the events occurring in auricle and ventricle were transcribed. In some experiments, the sounds were withdrawn from the ventricular cavities and were allowed to rest in the aorta, thus registering both the arterial pressure, and the onset and velocity of the blood current in that vessel. An instrument that transcribes arterial records is called a sphygmograph. Among the first instruments for direct sphygmography in man was that constructed by Marey (Fig. 32). The instrument is strapped on the wrist. A button with its spring is placed over the radial; the pulsations of the latter are transmitted

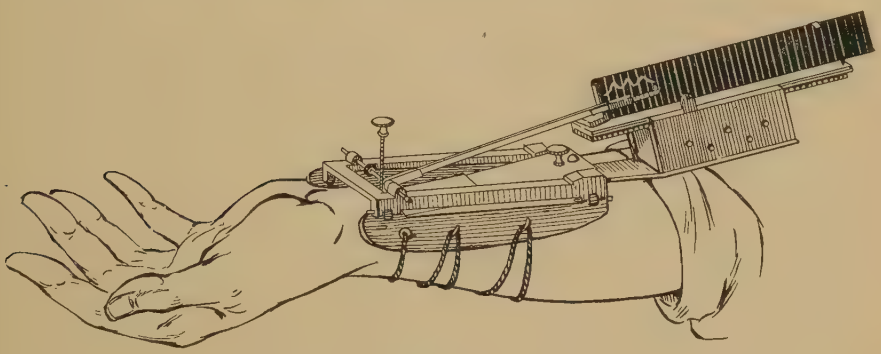

FIG. 32.—Marey's sphygmograph, illustrating an early type.

to a lever and transcribed upon a blackened surface. This instrument and the principle upon which it depended was later improved upon by Dudgeon, von Jacquet, Frank and Petter, Uskoff and others. The Uskoff sphygmograph transcribes the arterial pulsations more accurately than the older models because of the sensitiveness of its spring arrangement. For purposes of more accurate and detailed registration, Frank employed an optical device, a segment capsule as the recording member. The capsule is not circular but has an oblated, straight side forming the chord of the circle. Over the capsule a rubber membrane is stretched. A small trapezoidal plate is glued upon the straight chord side of the membrane. Upon this plate, at its free edge, is cemented a tiny mirror. The entire segment capsule with its mirror is so placed that it can readily be moved in a vertical and horizontal direction by thumb screws. The light is supplied by an arc or incandescent light which throws its beams upon the mirror of the capsule by means of a focusing lens. The light is then reflected from the mirror upon a moving photofilm apparatus, which is supplied with a motor for turning, and a shutter for exposing, the film. Each capsule has its separate focusing

lens so that various tracings (arterial and venous) may be simultaneously reflected and photographed. When used with a wrist sphygmograph, the segment capsule is put in circuit with the wrist pelotte and tambour by means of elastic tubing. Thus connected, the radial pulsations are trans-

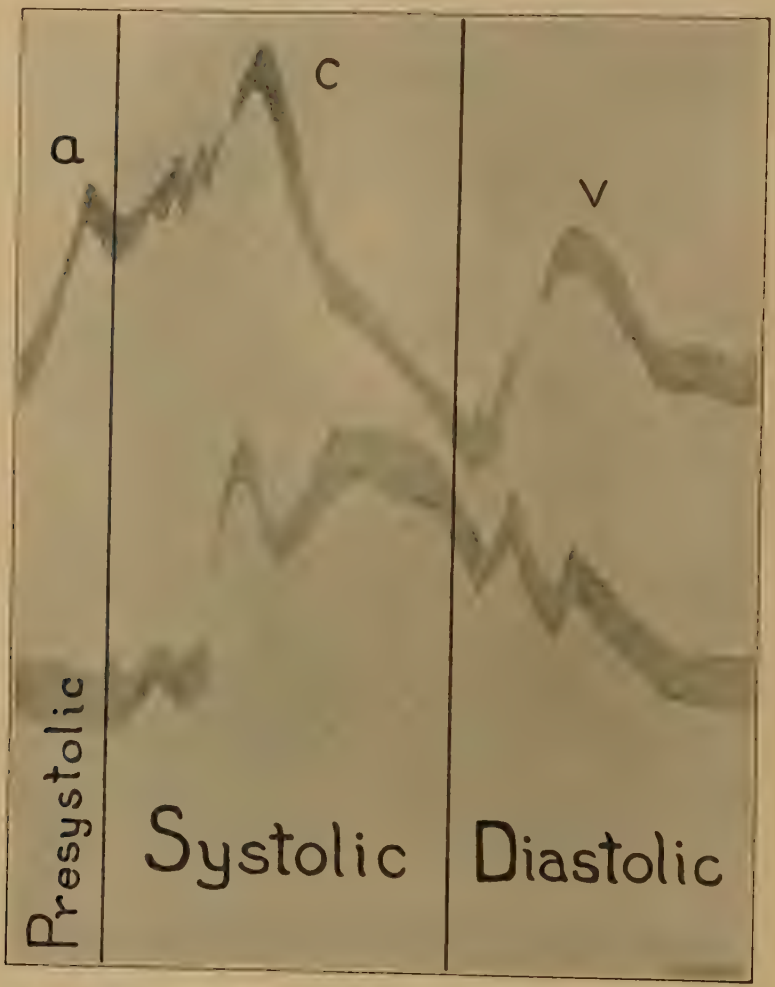


FIG. 33.—Upper tracing, supraclavicular venous pulse. Lower tracing, subclavian pulse. The letters *a*, *c* and *v* have the same significance as in the ink polygraphic tracings to be described later. (*After Wiggers.*)

mitted to the mirror capsule, the reflections from which are photographed as just indicated. In this manner, very accurate arterial and venous pulse tracings may be obtained (Fig. 33). The chief objections to the common use of the Frank mirror capsule are its intricacy and the time required for photography.

In addition to the sphygmographs already alluded to, many others have at various times been used. Brief mention may be made of the onychograph (used on the nail bed), the tachograph (depending on the variations of a gas flame, placed in circuit with the pulse by tubing), the capillary sphygmograph, the sphygmopalpeur, the sphygmoscope: The names are sufficiently descriptive to explain their special applications and uses. Tracings may also be made by introducing a sound in the esophagus, by which means pulsations of the left auricle can be transcribed.

Instruments that simultaneously transcribe arterial, cardiac and jugular pulsations are called polygraphs. For clinical use I have found the Mackenzie

FIG. 34.—Mackenzie ink polygraph.

Cl, Clockwork; Ti, time-marker; S, screw which regulates speed of time-marker; S.B., 1 and 2, keys for winding the driving gear and time-marker; Le, lever for starting and stopping the driving mechanism; Sl, slot for support of the paper roll (P.R.); Ar, arm for the support of the two transmitting tambours to the receiving apparatus; P, writing pens; E.T., elastic tubing connecting the transmitting tambours to the receiving apparatus; Cu, cup for receiving venous or cardiac pulsations; L.S., leather strap for buckling over the radial artery; Bu, button for receiving the radial pulsations; Sp, screw for regulating the pressure of the button upon the radial; W.T., wrist tambour.

Ink Polygraph satisfactory. Its compact size, the comparative simplicity and ease of operation, the fact that if necessary very long records can be taken, and the use of ink and a paper roll instead of smoked paper, make it suitable for clinical work.

The Mackenzie polygraph (Fig. 34) consists essentially of a clock work (Cl) encased in a metal container. The clock is fitted with a time-marker (Ti) which ticks and marks fifth seconds by means of a small pen. The speed of the time-marker is regulated by a small screw (S). There are two

separate keys (S.B.1, S.B. 2), one for winding the driving gear, the other for the time-marker. There is also a small lever (Le) which starts and stops the driving mechanism. Attached to one side of the case is a slot (Sl) into which is fitted a support for a paper roll (Pr). On the opposite side is a smaller slot into which fits a long, narrow arm (Ar) for the support of two transmitting tambours (TT); the latter are so arranged that they can move in any direction. To the tambours are attached long writing pens (P); the pressure of the points upon the paper may be regulated by manipulating the tambours. Elastic tubing (ET) connects each tambour separately with the receiving apparatus that is placed over the venous and arterial pulse in order to transcribe their pulsations. The receiver for the jugular is a small circular metal cup (Cu): This may also be used for registering cardiac pulsations by placing it over the apex. For transmitting radial pulsations, a perforated leather strap (LS) is buckled about the wrist and so adjusted that the button (Bu) or pelotte rests upon the most prominently pulsating part of the radial; the pressure of the pelotte upon the latter is regulated by a small flat spring (Sp). There is a broad wrist tambour (WT) which rests upon the button and transmits radial pulsations to the writing pens through the transmitting tambour.

**Method of Use of the Mackenzie Polygraph.**—The polygraph is used as follows: The driving mechanism and time-marker are first wound, the paper roll is set in place, the pens thoroughly inked and lightly adjusted upon the paper. After palpating the radial, its most pulsatile point is marked by an ink spot, or preferably by two rectangular lines (|_), one along the radial, the other across the wrist; these serve as guides for the proper position of the pelotte and wrist strap. The best position of the wrist is with the hand in moderate extension or hyperextension, because this tends to make the radial artery more superficial. This position can be conveniently maintained by firmly pressing the extended hand against some stable object like a table. The upper strap of the wrist attachment is put on loosely so as not to obliterate the artery; the lower is buckled on firmly. The spring regulating the pressure of the pelotte is then pressed down sufficiently to make the latter bob vigorously with the radial pulsations. The wrist tambour is slipped in position with its screw support loose, so that the metal tip on the under surface of the tambour rests fully upon the bobbing button; it is then screwed and held in this position. Thus through the receiving and transmitting air system of tambours and their connecting elastic tubing, the radial pulsations are transmitted to the writing pens.

To transmit and transcribe venous pulsations the metal cup is placed over the jugular bulb (Fig. 35), preferably on the right side, because the vein is usually more prominent on that side. The neck of the cup is grasped between the fore and middle fingers, the rim by the thumb, and the cup slid along the outer border of the sterno-mastoid muscle until it touches the clavicle. It thus rests over the triangle area formed by the jugular vein (with its

bulb), the inner end of the clavicle, and the sterno-mastoid muscle. The patient is made to lie as flat as possible; he should breathe quietly, for stertorous breathing interferes with proper registration. Rigidity of the neck muscles also mars pulsations. Superabundance of fat and respiratory dyspnoea are other factors which may interfere with or vitiate accurate registration.

After the wrist tambour and venous cup have been satisfactorily adjusted, the pens are separately slid across the paper, so as to establish vertical lines

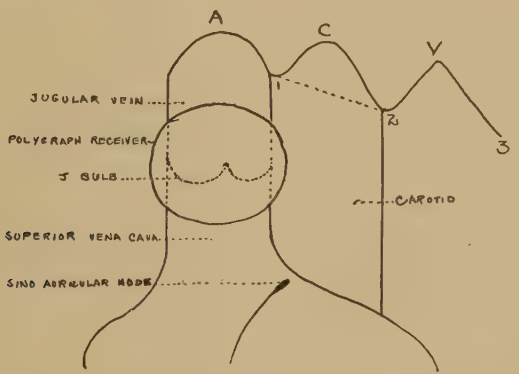


FIG. 35.—Schematic view of the *a–c–v* waves and of the jugular bulb.

(coincident ordinates) for measurement of the curves. These lines need not necessarily be continuous, for simultaneous venous and arterial pulsations may be measured off and standardized by means of calipers. The driving mechanism is now released, and arterial and venous pulsations simultaneously registered.

As already stated, early experimentors gained information regarding auricular pressure curves by inserting sounds directly into the auricles of dogs and horses; they found that auricular contractions were accompanied by increased auricular pressure, that is, by positive pressure waves (Figs. 36, 57). While there is a general correspondence between such pressure curves and human jugular tracings obtained by the polygraph, it must be remembered that the latter tracings primarily depend upon differences in *volume* created in the confined air space of the cup resting over the jugular bulb. These volumetric differences are transmitted to the tambour and the pen is then correspondingly deflected. Hence differences in auricular *pressure* are not necessarily transmitted and transcribed as corresponding differences in the volumetric waves by the cup over the jugular bulb; and as a corollary, one can rarely predicate and estimate auricular pressure by the excursion and direction of the "venous" polygraphic waves.

**The Waves of the Normal Jugular Pulse. The Normal Phlebogram.** Frequent observations have shown that, corresponding to the first auricular pressure curve coincident with auricular systole, there exists normally

a venous pulse beat seen in the venous trunks at the root of the neck. In man, for each radial beat, there are in the normal jugular tracing three waves or elevations; each elevation is accompanied by a corresponding depression (Fig. 37). We shall follow the simple nomenclature usually adopted in

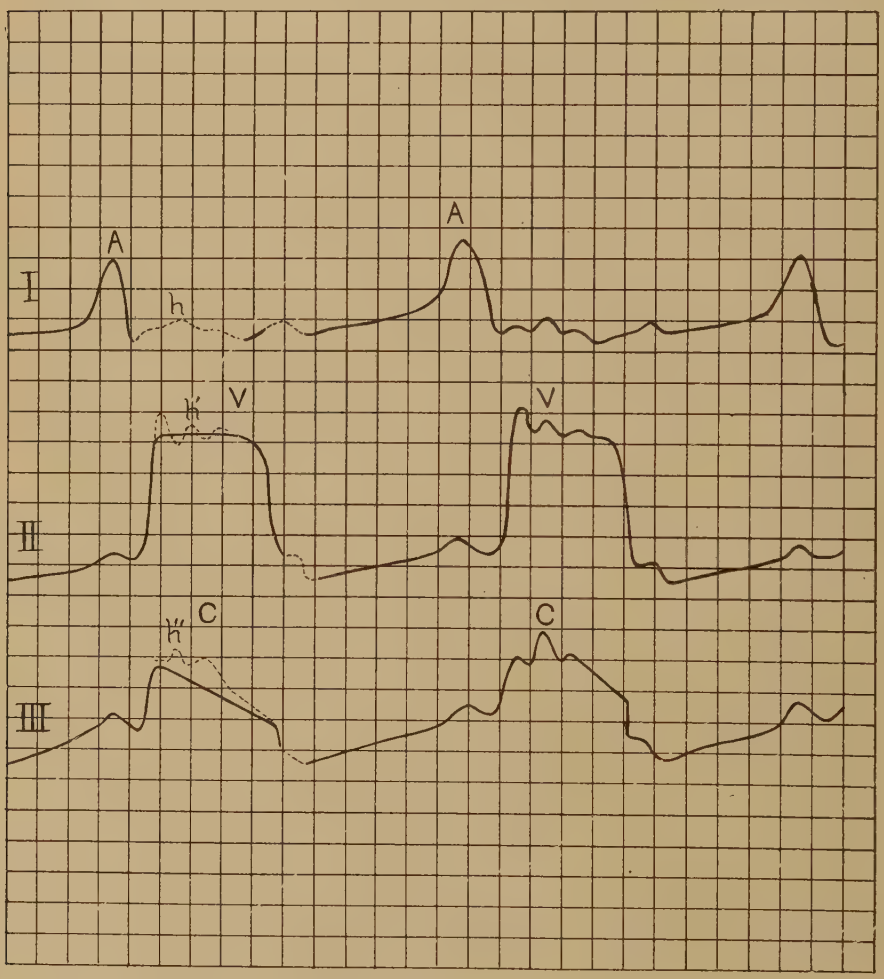


FIG. 36.—Simultaneous tracings from the right auricle (I); the right ventricle (II) and systolic shock of the ventricle (III) of the horse; *a*—auricular systole; *v*—ventricular systole; *c*, ventricular shock. According to Marey, the smaller undulations *h*, *h'*, *h''* are due to closure of the auriculo-ventricular valves; the vertical divisions measure $\frac{1}{10}$ seconds. (*After Marey.*)

the literature and call the elevations the *a-c-v*. waves. The *a* refers to the auricular, *c* to the carotid, and *v* to the ventricular filling wave. The rise (Figs. 35, 37) and fall (Figs. 35, 37) of the first wave *a* are caused by the reflex wave produced in the veins of the neck by auricular systole. In rhythmically

beating hearts, the *a* wave comes before the advent of ventricular systole (*c* wave). Absence of the *a* elevation in those types of arrhythmia in which experimental and electrocardiographic evidence shows absence of normal rhythmic auricular contractions (auricular fibrillation, Chapter X), and abnormal position of the *a* wave in arrhythmias affecting relationship between auricular and ventricular contractions (heart block, Chapter X)—these constitute confirmatory evidence that the *a* wave is in the main, if not entirely, due to auricular systole. If the jugular tracings corresponded to the experimental auricular pressure curves, they would show but two waves, that is, the *a* wave marking auricular systole, with its fall (Fig. 37, 1-2) and the *v* (ventricular filling) wave with its fall (Fig. 37, 2-3). The post-auricular fall, however, is interrupted by the advent of another elevation, the *c* wave. For the present disregarding the latter, the chief cause of the post-auricular fall of pressure (Figs. 35, 37 1-2) is undoubtedly auricular relaxation following auricular systole; ventricular systole influences and increases this relaxation mainly by dragging down the interventricular septum, and to a lesser degree by producing diminished intrathoracic pressure. The physiological limit of the duration of the *a* wave as measured at its base line is one fifth of a second.

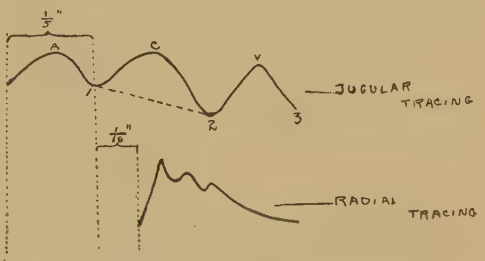


FIG. 37.—Diagrammatic representation of the normal polygraphic curve.

*a*, auricular wave; *c*, carotid wave; *v*, ventricular filling wave.

The cause of the carotid (*c*) wave is still a matter of dispute. Its occasional appearance one twentieth of a second before the onset of carotid pulsation, and its presence after experimental ligation of the carotids have been offered as evidence that the *c* wave is not due to carotid pulsation. However, those who have worked with the cup receiver over the jugular bulb will have observed how the venous tracing is often vitiated by placing the receiver too close to the carotid, in consequence of which the usual polygraphic *c* wave will often obtrude itself upon the venous tracing. Although there exists some dispute as to its cause, the practical importance of the incidence of the *c* wave in the study of the human phlebogram rests upon the fact that its foot point (its beginning) is coincident with the onset of carotid pulsation. The determination of the foot point of *c* therefore, becomes an important landmark in the study of the polygram. Since the pulse wave reaches the wrist approximately one tenth of a second after its arrival at the carotid artery, and since the onset of the individual radial beats is readily discernible in the arteriogram, the foot point of the *c* wave may be determined and distinguished from the *a* and *v* waves in the phlebogram by measuring with calipers from coin-

cident ordinates in the radial and venous tracings. These ordinates are derived by stopping the driving mechanism for a moment and sliding both pens across the paper (Fig. 38). They need not necessarily form a continuous line; in fact, they rarely do because the pens are often not in alignment. Calipers are then taken to measure from the ordinate to the foot point of any radial beat to the left of it. This caliber distance is then transferred to the left of the ordinate in the venous tracing. The foot point of the *c* wave will be one tenth of a second to the left of this point, for this marks the difference in time between the onset of the carotid and radial beats. The width of the one tenth second is readily derived, because the time marker marks and spaces one fifth seconds. With the *c* wave determined, it becomes a simple matter in good curves to determine the *a* and *v* waves when the pulse beats rhythmically: for the pre-carotid is the *a* and the post-carotid, the *v* wave.

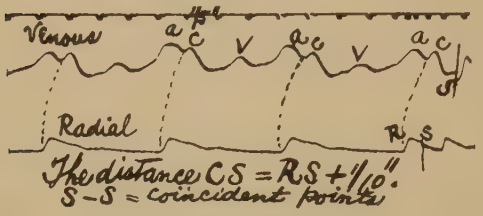


FIG. 38.—Normal venous and radial tracing showing rhythmical *a–c–v* waves in the jugular tracing, and regular radial beats. The points S–S are coincident ordinates derived by sliding the pens when the driving mechanism is at rest. The method of derivation of the foot point of the *c* wave is also shown.

The third positive wave (Figs. 35, 37) *v*, the ventricular wave which follows the second depression, is due to ventricular systole; it results almost entirely from reflux of the stored auricular blood being forced into the venous system during ventricular systole while the auriculo-ventricular valves are still closed. Another factor in the production of the *v* wave is sudden release of the base of the ventricle at the commencement of ventricular diastole. The slightly variable beginning of the *v* wave depends upon the varying amounts of blood stored up during auricular diastole. Its termination is coincident with the opening of the tricuspid valves; hence it, in conjunction with the *a* and *c* waves, form certain definite landmarks of the time events of the contracting chambers.

The third fall or depression (Fig. 37, 3) is caused by the rapid drop in pressure which follows the beginning of ventricular diastole and the opening of the auriculo-ventricular valves.

By translating the knowledge gained from the orderly sequence of auricular and carotid pulsations (the *a* and *c* waves) as transcribed graphically, into terms of auricular and ventricular systoles, we have the means of studying rhythmical sequential cardiac cycles—the normal pulse and phlebogram—as well as the numerous disturbances of rhythm that may affect auricles and ventricles.

**Variations in the Normal Phlebogram.**—There are certain variations in the normal phlebogram which requires consideration. The *a* wave is occa-

sionally bifurcated at its apex, *i.e.*, at the height of auricular systole; such splits may possibly result from a venous reflux wave produced by a sharp flapping action of the valves in the jugular bulb. The *c* wave is sometimes split (Figs. 39, 40, 41). When this occurs, the first part is usually high and its fall sharp. It is due to the fling of the lever caused by placing the cup too close to the carotid, or to causes similar to those of the dicrotic notch of the

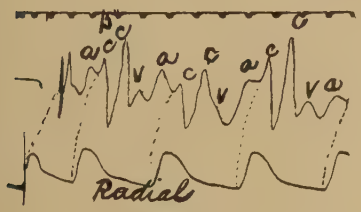


FIG. 39.

FIG. 40.

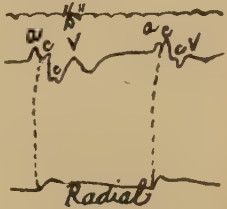


FIG. 41.

FIGS. 39, 40 and 41.—These figures show a split *c* (*c*–*c*) wave in the venous tracing.

radial (q.v.). Another variation of the phlebogram is the occasional blending of venous and arterial tracings; the *c*-*v* waves then resemble an arteriogram (Fig. 42). The *v* wave is sometimes divided; this division is probably caused by the two factors in its production acting somewhat asynchronously.

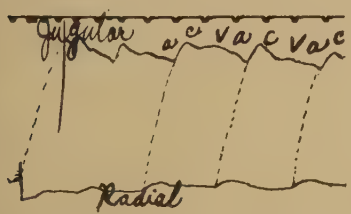


FIG. 42.—Combined venous and arterial tracing in the jugular.

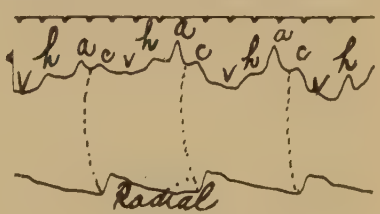


FIG. 43.

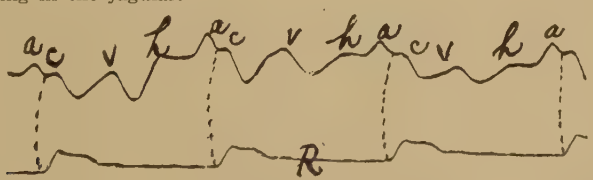


FIG. 44.

FIGS. 43, 44.—Phlebograms showing *h* waves.

Another wave, the so-called *h* wave is sometimes seen in middiastole or directly preceding the *a* peak (Figs. 43, 44), especially when the heart beats slowly. It is usually regarded as a reflux wave due to the rather sharp ballooning of the right ventricular tricuspid valve from the rush of inpouring blood during diastole. It has also been regarded by some as related to the third heart sound. The *h* wave, however, may be present even when there is no electrocardiographic evidence of a third heart sound (Chapter XIII).

**Limitation of Inferences from the Phlebogram.**—With reference to information derived from the normal rhythmic tracing, it must be emphasized

that, because of the mechanical limitations of the polygraph, because the waves measure *volumetric* (*not* pressure) changes, and because of the manner of application of the venous cup, only very rarely can conclusions regarding auricular or ventricular energy be drawn. I have, for example, taken many

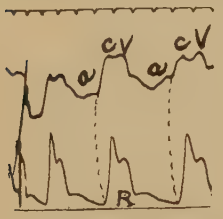


FIG. 45.—Normal phlebogram from a case of aortic regurgitation.

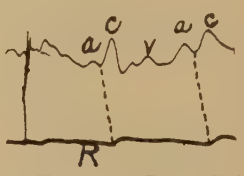


FIG. 46.—R, radial tracing. Normal jugular tracing from a patient with aortic stenosis and a double mitral lesion.

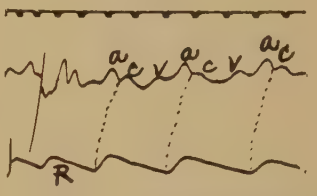


FIG. 47.—Normal jugular tracing from a patient with aortic stenosis and a double mitral lesion.

venous tracings from patients with compensated and decompensated valvular and myocardial lesions, and after comparison with normal tracings, I have been unable to discover any distinction between them. Figures 45–52 taken

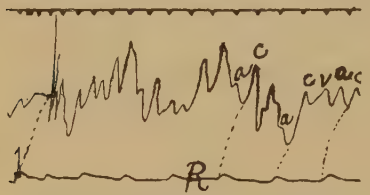


FIG. 48.—Normal sized *a* and other peaks from a case of mitral stenosis.

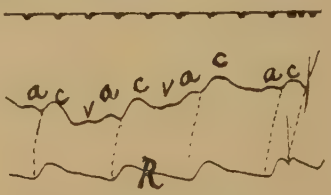


FIG. 49.—Normal *a* wave from a case of mitral regurgitation.

from patients with various diseases illustrate this fact. Another important consideration which applies particularly to interpretation of arrhythmias from the phlebogram is that the waves in the tracing must be clear and

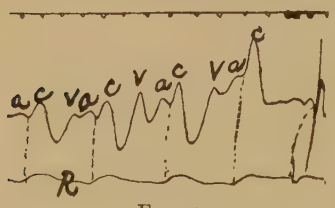


FIG. 50.

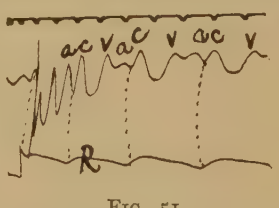


FIG. 51.

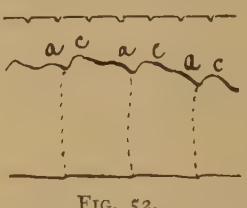


FIG. 52.

FIGS. 50, 51, and 52.—Normal jugular tracings from cases of exophthalmic goiter.

distinct; otherwise the diagnosis of arrhythmias becomes to a great extent a matter of hazard.

**The Radial Pulse.** — Because of the method of instrumental application —a spring and pelotte pressing upon the radial artery—the arterial sphygmogram (arteriogram) represents only crass differences of arterial pressure. The amount of spring tension required to sufficiently occlude the artery for

the purposes of tracing is quite variable, hence the resultant curve is in many instances neither an accurate nor an approximate measure of the amount of arterial pressure. In addition to these mechanical drawbacks, extraneous factors, *e.g.*, the position of the radial, its accessibility, the pliability of its walls, etc., are considerations which profoundly modify and mar inferences drawn from the pulse tracing; hence doubtless the confusion, the differences of opinion and the varied interpretations drawn by clinicians from arteriograms. The special value derived from the radial tracing in the polygram

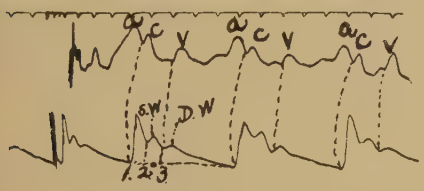


FIG. 53.

FIG. 54.

FIGS. 53, 54.—Normal radial pulse tracings.

1–2, Abrupt rise probably due to instrumental fling of the lever (also called primary or percussion wave); 1–3, time of ventricular systole, the aortic valves are open; D.N, dicrotic notch; D.W, dicrotic wave; S.W, systolic wave (also called tidal and predicrotic wave).

rests upon knowledge gained regarding cardiac rate and rhythm, and in the aid it gives in fixing the time relation of events in the cardiac cycle; for, as already noted, the foot point of the radial serves as a standard for determining the incidence of the *c* wave in the venous tracing.

The first elevation of the radial beat (Figs. 53, 54), usually called the primary or percussion wave, is generally steep; its fall, sharp. It is due to the sudden instrumental fling given to the pelotte and lever by the sharp impact of the onrushing blood. It occurs in, and is part of the wave produced by systolic arterial distension, and may be regarded as an initial artificial peak superimposed upon the arterial wave during systole. It is immediately followed by the systolic wave, sometimes called the secondary tidal or predicrotic wave (Figs. 53, 54, S.W. 2, 3). The degree of the fall of systolic pressure—graphically represented in the tracing by the angle of slope and suddeness of fall of the wave—depends upon the difference between the systolic and diastolic pressures. This difference which can be measured by a blood pressure apparatus (Chapter XXV) is called the pulse pressure (Chapter XXV). If this be large, the down slope is apt to be steep; if small, it becomes a gradual one. The termination of the systolic, and the beginning of the dicrotic wave (Figs. 53, 54, D.W.) is marked by the dicrotic notch (Fig. 54, D.N.). This notch corresponds to certain events in the cardiac cycle: The end of ventricular systole and the foot point of the *v* wave in the venous tracing (Fig. 53). Other small waves are sometimes found in the radial tracing; their cause and significance are not known.

The cause of the dicrotic wave is still in dispute. Mackenzie regards it as due to sudden relaxation of the ventricular wall, including that portion supporting the aorta. According to him there is thus developed a tendency

to the production of a negative aortic wave, which is checked by the sudden stretching of the membranous aortic valves, thereby causing a second positive—the dicrotic wave. It has been experimentally demonstrated in a circulatory model in which the arterial system is represented by elastic tubing, that sudden check of the inflow produces a suction or negative pressure behind the column of fluid; as a consequence, there are resultant waves. Indeed, "dicrotic" waves have been produced in an "arterial system" in which the pumping mechanism was a syringe not comparable to the heart, a fact showing that these waves may be entirely the result of pressure effects in elastic arteries. With the influx of fluid, the tube expands; with the sudden cessation of the flow, the resultant negative pressure in a rigid tube would lead only to a reflux of fluid. In elastic tubing, however, represented in the human being by the aorta, there is the additional force of elastic recoil. Both forces—suction and elastic recoil—produce shrinkage beyond the natural caliber of the tube (aorta); the elastic, constricted tube causes secondary expansion, and with it, the secondary pulsatile "dicrotic" wave. These physical facts applied to the heart, in addition to sudden ventricular relaxation following systole, may account for the dicrotic wave in man.

It has also been held that the dicrotic wave is "reflected" from the periphery. Under such circumstances, the distance between dicrotic and primary crests ought to diminish as the arteries recede in distance from the heart, and there should be no dicrotism in the proximal part of the compressed artery. These suppositions, however, are not borne out by arterial tracings which have been taken under such conditions. In addition, the manifold arterial division at the periphery would appear to make *one* large "reflected" wave improbable.

The height of the dicrotic notch is ordinarily about one half that of the systolic wave. In cases with sharp fall of arterial pressure following ventricular systole, the notch is abnormally low. This happens frequently in

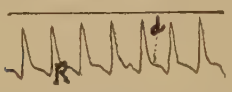


FIG. 55.—Low dicrotic notch (*d*) from a case of aortic regurgitation with decompensation.

FIG. 56.—Low dicrotic notch (*d*) from a case of aortic aneurism with cardiac failure.

aortic regurgitation with cardiac failure (Fig. 55), but it is also found in other decompensated heart lesions, valvular or myocardial in origin (Fig. 56), especially when the pulse pressure is large.

**Types of Pulse.**—From what has preceded, it is evident that only exceptionally can definite conclusions regarding the "strength" or "weakness" of the circulation be drawn from graphic records. In fact, the terms "weak" and "strong" pulse are usually misapplied to what should properly be called "soft" and "hard," respectively. The old terms previously in use had the advantage of describing the physical impression given to the examining

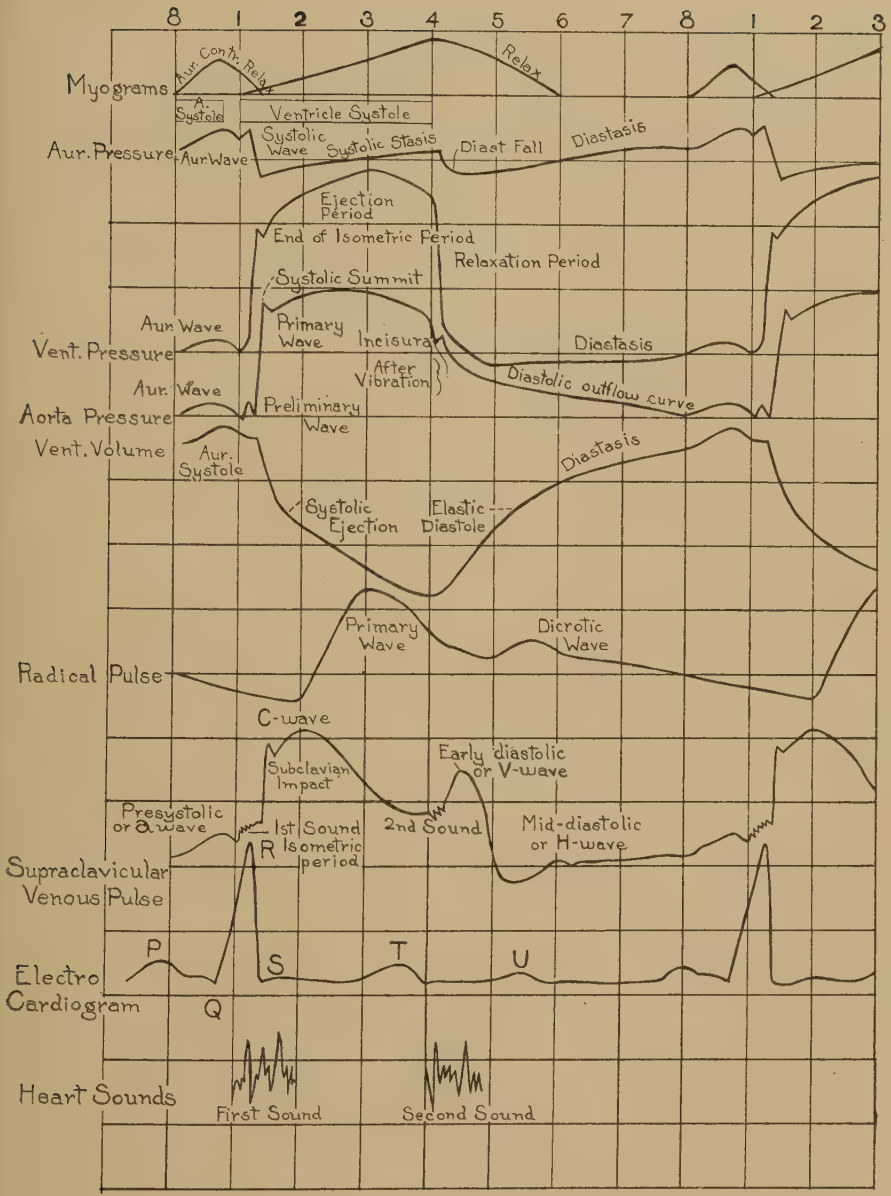


FIG. 57.—Simultaneous events in cardiac cycle. (*After Wiggers.*)

finger. When thus *descriptively* applied, their limitations understood, and no deductions regarding the state of circulation drawn from them alone, the old terms serve a useful purpose. These terms and their definitions are as follows: A sudden fall of the pulse wave produces what is known as the "collapsing" pulse; if extreme, it becomes the typical "water hammer" or Corrigan pulse. The radial pulse can also be described as large and expansile (*pulsus magnus*), small or compressible (*pulsus mollis*), or hard and incompressible (*pulsus durus*). The rise of the pulse wave may be quick (*pulsus celer*) or slow (*pulsus tardus*). If dicrotism is sufficiently marked to be felt in taking the pulse, it is known as dicrotic; if the dicrotic notch breaks low and the dicrotic wave is marked, it is called hyperdicrotic. Occasionally, the pulse wave feels unduly sustained at the point of its maximal pulsation and falls slowly—the anacrotic pulse. This is commonly regarded as diagnostic of some form of compression of the aorta; it has, however, also been found as a result of peripheral factors alone (Lewis). The bisferiens gives the sensation of a double pulsatile impact: It is produced by the rather equal split of the systolic plateau by the predicrotic or instrumental wave.

Important information of the condition of the radial artery itself is sometimes gained by careful palpation. Marked nodosity, thickening, and tortuosity are immediately apparent. The opposite information—that of a normal elastic arterial wall—is not so readily derived. Examination is best practiced by emptying the artery and stripping back the blood with the finger of one hand and palapting the collapsed radial with the other hand. The radial, if normal, will be barely definable as a separate strand; if the radial be thickened and its walls stiff, the emptied artery is distinctly palpable below the point of compression.

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## CHAPTER VIII

## THE ELECTROMETER—THE ELECTROCARDIOGRAPH—ELECTROCARDIOGRAMS

Records obtained from the use of the capillary electrometer and of the electrocardiographic apparatus are based upon the fundamental physiological fact that any muscle in contracting produces a definite although minute amount of electricity.

**Capillary Electrometer.**—The capillary electrometer consists essentially of a capillary tube containing mercury; the tube dips into a weak solution of sulphuric acid. If the electrical reaction of a contracting muscle is to be tested, one end of the muscle preparation is placed in circuit with the mercury, and the other end with the sulphuric acid solution. The muscle is thus connected with the capillary electrometer by means of these electrodes. If then, as a result of muscular contraction, an electric current passes through the electrometer, the mercury in the capillary tube is displaced in the

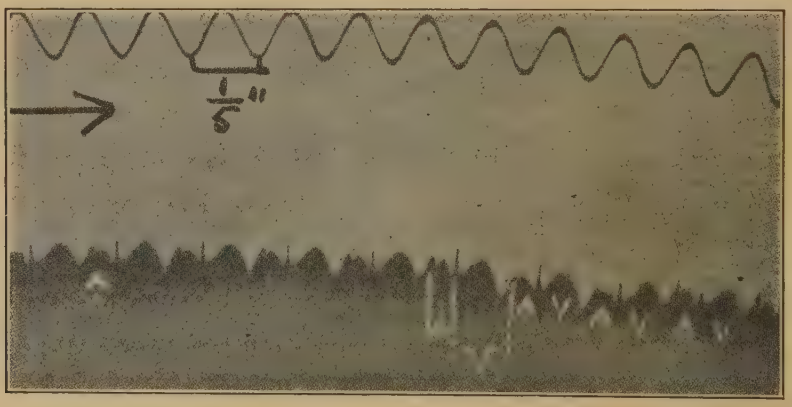


FIG. 58.—Electrometer record of changes in the rabbit's heart with one contact on the right auricle and the other on the apex of the right ventricle. A = auricular, V = ventricular beat. The heart rate is 250 per minute. Upward movement signifies relative negativity ( = muscular activity) at or near the auricular contact. (*After Gotch.*)

direction of the induced current. The motion of the mercurial column can then be photographed by an appropriate lighting and photokymographic apparatus. The resultant graphic record (Fig. 58) therefore expresses muscular activity in terms of changes in the mercurial column. The electrometer does not possess the accuracy or sensitiveness of the electrocardiograph, which, where available, has become the standard modern instrument for

measuring and transcribing the electrical reaction produced by the living heart.

**The Electrocardiograph or String Galvanometer.**—There seems to be a current impression among physicians not especially interested in the field of cardiology that the principles of electrocardiography are too intricate to grasp; this feeling also applies to electrocardiograms and their interpretation. I believe that the fault is at least partly due to the cardiologists themselves, for their description of the apparatus and of the resultant graphic curves (electrocardiograms) is often highly technical, while the underlying facts, in themselves simple and easily grasped, are usually lost or become confused in a maze of unnecessary technical detail. I therefore wish to emphasize that the practitioner can readily understand the workings of the electrocardiograph and the interpretation of electrocardiograms, if he will but grasp the few underlying simple electro-physiological facts, some of which have already been briefly alluded to. The entire field of electrocardiography with its many symbols will then become bereft of much of its complexity. As in many other new sciences, there are indeed still many debatable points regarding the causation and interpretation of various deflections and curves, but for clinical purposes much of the disputed ground can be discarded.

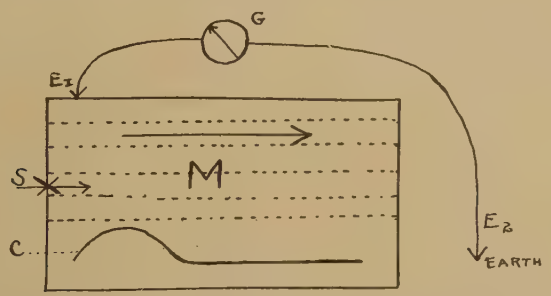


FIG. 59.—Scheme of monophasic action current. (*Modified after Kraus and Nicolai.*) M, Muscle consisting of parallel fibers; S, point of stimulation; E1, non-polarizable electrode; E2, earthed electrode; G, galvanometer; C, resultant monophasic curve.

It is a well established electro-physiological fact that a minute amount of electricity is caused by any muscle during its stage of contraction. The electrical current thus produced, if allowed to pass through a sensitive galvanometer, causes deflection of the needle. For example, if a muscle consisting of parallel fibers (Fig. 59, M) be stimulated at the point S, the surface at the area connected by means of a non-polarizable electrode (E1) to a galvanometer (G), and the other electrode (E2) grounded so that it remains constant (equipotential), then the muscle at S in contracting becomes electrically negative relative to the remainder of the musculature. Hence negative electricity and muscular contractibility are equivalent and synonomous. As the current passes, the stimulated ends become electrically quiescent; hence the electropotential falls to and reaches zero, at the same time

that the needle of the galvanometer has been deflected and comes back to rest at zero. If recorded, such deflection would be a monophasic curve (Fig. 59). If, instead of being grounded, that electrode is connected with the other end of the same parallel fibered muscle (Fig. 60, E2) and the latter is then stimulated at the point S (Fig. 60), the stimulated area in contracting again becomes electronegative, while the other areas of the muscle remain electropositive because they are not yet in contraction. With the beginning of the flow of the electric current the needle of the galvanometer starts its deflection. When the wave of muscular contraction arrives at the center of the muscle, the needle does not move because this zone now draws negative electricity in equal amounts from both sides. With the passage of the contraction wave toward E2, the latter becomes electronegative, the current flows from E2 to + E1 and the needle is deflected in the opposite direction. In other words, with each change of electrical signs, the needle is deflected, and in the assumed instance of a parallel-fibered muscle, a diphasic curve results.

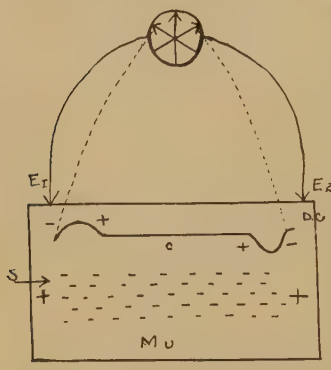


FIG. 60.—Scheme of diphasic action current. (*Modified after Kraus and Nikolai.*)

Mu, parallel fibered muscle; C, center point; S, point of stimulation; E1, E2, nonpolarizable electrodes. Minus signs over Mu show that that area has become electronegative when reached by the contraction wave, and that both ends are then temporarily positive. (See text.)

In view of later electrocardiographic considerations, it is important to emphasize, as evidenced by the above simple experiment, that the resultant curve depends upon the direction of a contraction wave in the muscle and upon the point at which the contraction arises. It further depends upon the axis of the muscle mass; an oval or an irregularly shaped muscle, for example, will not give an equally balanced diphasic curve in contracting because the electrical waves themselves would not be equally balanced, so to speak. It is also evident that the course of the contraction wave, of the galvanometric deflections, and of areas of relative electrical negativity are intimately correlated. Finally, it is important to remember that, as already stated, a muscle at the moment of contraction is electrically negative, so that muscular activity and electrical negativity are practically interchangeable terms. These simple considerations regarding the action of a contracting muscle upon a galvanometric needle will serve to clarify the principles underlying the electrocardiographic apparatus.

It was chiefly through the work of Einthoven that the electrocardiographic apparatus (Figs. 63, 64) reached its present stage of perfection as a laboratory and clinical instrument. The standard instrument consists essentially of a fine conducting fiber scarcely visible to the naked eye, 2 to 4 microns (0.002 to 0.004 m.m.) in diameter, that lies in a narrow space between

two approximated poles of a powerful electromagnet (Figs. 61, 62, 63). The fiber or string, as it is usually called, is deflected by currents induced in it. These movements are magnified by a microscope resting in the magnetic pole shoes; the magnified movements are photographed by a moving photo-

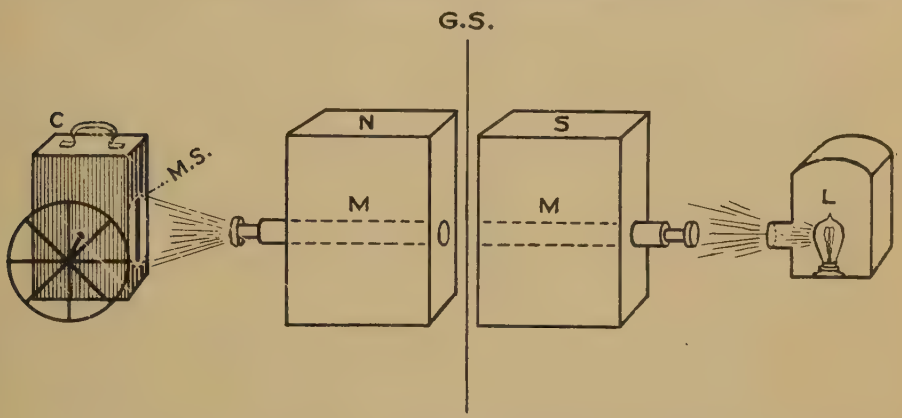


FIG. 61.—Schematic view of electrocardiographic apparatus.
N.S., north and south poles, respectively, of electromagnet; G.S., galvanometric string; M, microscope set in the electro-magnet; L, electric bulb light; C, camera containing film fed by motor; M.S., magnified image of string.

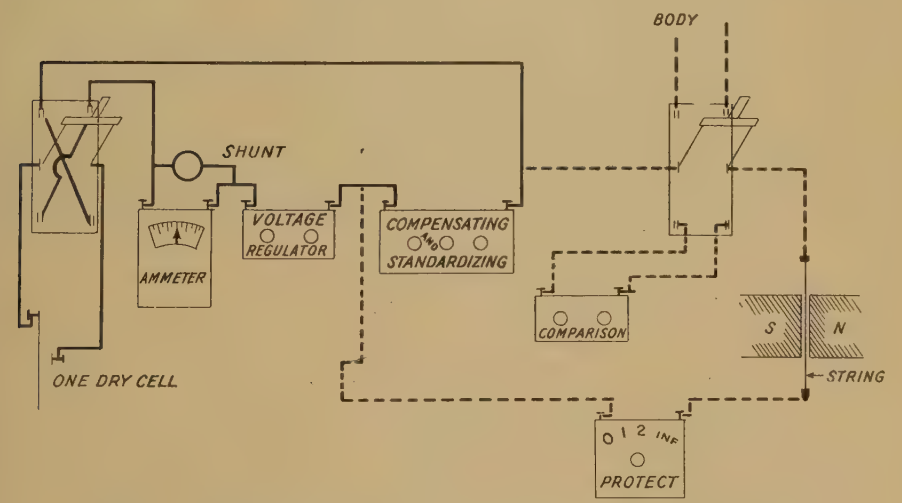


FIG. 62.—Scheme of rheostats and connection with the galvanometer.

film apparatus. The films, when developed, constitute the electrocardiograms. The string is usually made of an exceedingly fine quartz fiber coated with silver. The silver gives the conducting surface. Some strings are doubly coated with both silver and gold: This adds to their toughness and durability. The resistance of the string varies from 1500 to 7000 ohms. The

electrocardiographic apparatus which I use—the standard type—is schematically shown and described in Figs. 61 and 62 respectively. Figures 63 and 64 show the apparatus set in position. Figure 64 shows how the patient is placed in circuit with the apparatus. The electrodes used are described in the succeeding paragraph.

**Method of Taking an Electrocardiogram. The Three Leads or Derivations.**—In the human being, the indirect method of taking electrocardiograms must of course be employed. This is accomplished by various types of electrodes applied to the body: The current thus drawn off and flowing between the electrodes constitutes the "leads" or "derivations." There are

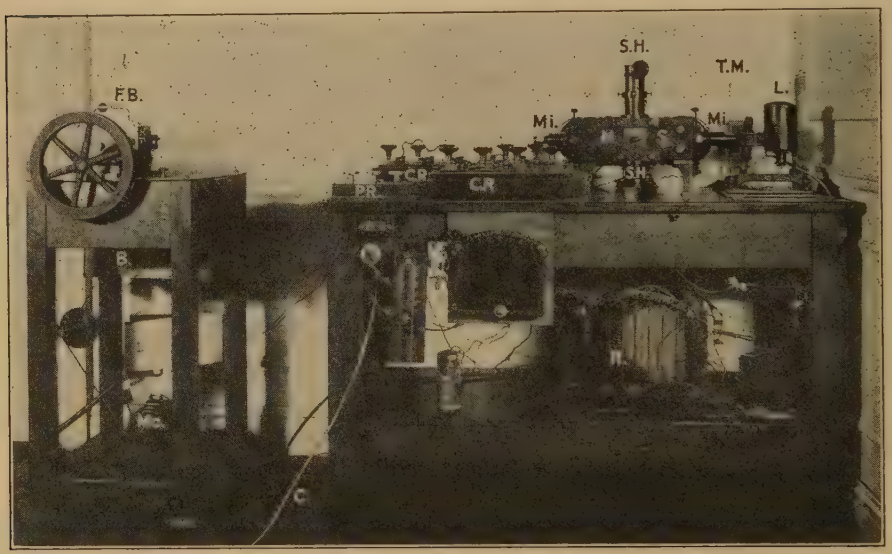


FIG. 63.—Photograph of the electrocardiographic Hindle-Williams apparatus in position. L, Electric bulb; P.R., rheostat for "leads" to patients; C, coil containing 3 "leads;" Mi, microscope; T.M., time marker; F.B., box containing roll of film; N.S., north and south poles of electromagnet; S.H., string house containing quartz fiber; Mo, motor for driving film; R, rheostat for storage batteries; C.R., crank rheostat.

various methods of employing non-polarizable electrodes to conduct the electric potential produced by cardiac activity to the galvanometer. One method consists in wrapping flannel bandages, each about six inches wide and 9 feet long, thoroughly soaked in a strong warm salt solution (6 oz. of salt to one pint of water), around each forearm and around the left leg of the patient. After these extremities have been covered by a few turns of the bandage, German silver electrodes with binding posts are included in its folds. These electrodes are 6 inches long and 5 inches wide, and are sufficiently thin and pliable to be bent and snugly applied. When patients can sit up, a simpler and more expeditious method consists in having three separate vessels containing strong warm salt-water solutions. In each

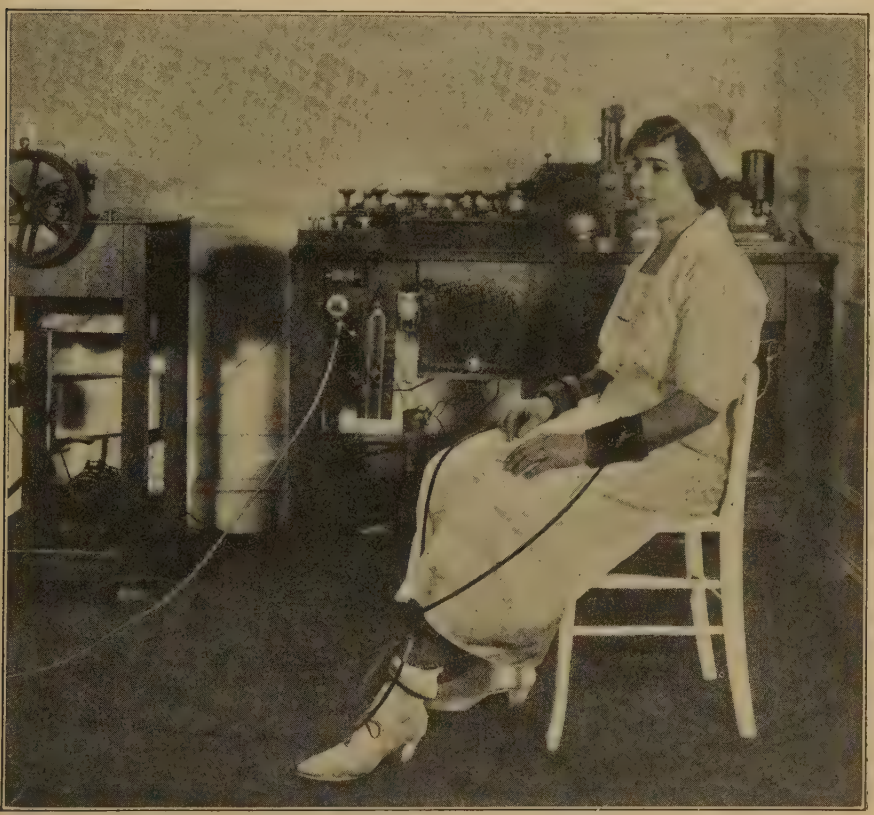

FIG. 64.—Electrocardiographic apparatus with a patient in circuit. The electrodes are fastened on right forearm, left forearm and left leg.

FIG. 65.—Lead foil electrodes devised by Robert Neubuck.

vessel is placed a porous cup containing a 100 per cent. zinc sulphate solution and a zinc plate with a binding post. Thus electrical connection is established between the patient and the electrocardiographic apparatus. In either case, binding posts are connected by wires distinguished by various colors, and are placed in circuit with the galvanometer by means of plugs and switches. More convenient, easier, as accurate, and giving no discomfort to the patient, are the lead foil electrodes recently devised (Fig. 65). The lead foil is made of an alloy of tin and lead (roentgen-ray protection foil) which is cut into strips approximately 9 inches long by 4 inches in width. Each is fastened by means of a brass screw and binding post to a rubber strip one half inch longer and wider than the foil strip. The forearm and left leg are washed and rubbed with a strong warm saline solution; a strip of gauze dipped in the same solution is then wound around these extremities, and the lead foil electrodes applied. As a convenient method of keeping the electrodes in place, broad rubber bands or pieces of tape may be used. These electrodes are also connected with the electrocardiographic apparatus in the usual way. I have also occasionally employed the same type of lead foil electrodes but cut into small one inch squares directly over the precordium in an effort to get the leads more directly from the corresponding underlying parts of the heart surface. In this manner it may be possible to take electrocardiograms in the human being somewhat comparable to the electrocardiograms taken with the leads placed in direct contact with the exposed hearts of animals.

Just as any other contracting muscle, so the heart in contracting, gives rise to waves of electric potential which spread from their source over the entire body. It is these that are conducted to the galvanometer by means of the non-polarizable electrodes which are wound around the patient's three extremities. There are three arbitrary directions of the current coursing through the heart that are employed in taking an electrocardiogram and that correspond to the three extremities. These are the so-called "leads" or "derivations." The current (Fig. 66) from the right to left arm (R.A.—L.A.), running chiefly across the base of the heart, is the first lead or derivation; that from the right arm (R.A.) to the left leg (L.L.), approximately parallel to the long axis of the heart, is the second, sometimes called the "strong" lead; that from the left arm (L.A.) to the left leg (L.L.) is the third lead and draws off the current coming mainly from the left side of the heart. To these I have occasionally added another lead by placing one electrode

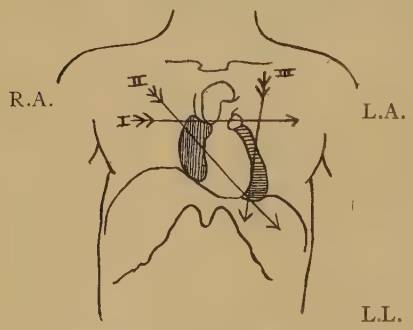


FIG. 66.—Diagram illustrating the three leads. R.A., right arm; L.A., left arm; L.L., left leg.

over the anterior surface of the chest and the other over the spine of the left scapula. For this purpose I place the "right arm" (R.A.) electrode anteriorly and the "left arm" electrode (L.A.) posteriorly, thus drawing off the electrical potential of the heart in an antero-posterior direction. I have not yet studied the subject sufficiently to discover whether the information thus derived is of added clinical value.

The photographic reproduction of the magnified deflections of the galvanometric string during the registration of the various leads constitutes the electrocardiogram. While the electrocardiograms of no two persons are exactly alike, and while physiological differences vary within wide limits, there is a general conformity to a normal type. A typical normal electrocardiogram is schematically shown in Fig. 67. It demonstrates the various waves or deviations and the approximate time required for each when the heart is beating rhythmically at the rate of 72 per minute. Each division parallel to the base line (the line of isopotential) is equivalent to one millivolt (one thousandth of a volt, ($10^{-4}$ volt) of current that is produced by cardiac contraction. The electrocardiogram, that is, the registration of the difference of electric potential, precedes the actual cardiac contraction by about .03 second.

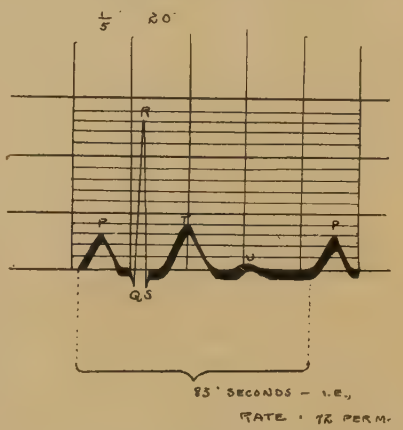


FIG. 67.—Schematic representation of a typical electrocardiogram, second lead. Each horizontal space measures one millivolt of current. The ordinates measure one-fifth second. The form and size of the various deviations are seen, as well as the time required for their formation.

**The Normal Electrocardiogram** (Fig. 69) will now be considered. The nomenclature I adopt is that first introduced by Einthoven and, with few exceptions, now in general use. Following the sequence of normal cardiac activity, the electrocardiogram is conveniently divided into that produced by auricular contraction—the auricular complex—and that by ventricular contraction—the ventricular complex. Animal experimentation, and observations on the human subject with normal and abnormal rhythms, especially those with heart block and auricular fibrillation, have confirmed the fact that the auricular complex (known as the P wave) is caused by auricular contraction. The P wave is normally directed upward and is usually somewhat flattened at its summit. After reaching the base line it is succeeded by a short horizontal isoelectric line, an evidence of quiescence of electric potential; that is to say, during this period, cardiac contraction has ceased and the heart is at rest. The ventricular complex consists of the QRST deviations; the chief of these are the R and T deflections. The Q and S vary in size; they are usually short, sharp peaks directed downward.

Upward deviations imply electrical negativity (*i.e.*, muscular contractility), at or near the base of the ventricles; downward deviations, at or near the ventricular apex. Hence the downwardly directed Q and S deviations imply that the base of the heart is at that moment electrically positive, (not contracting) and that the apical region consequently has the opposite electric sign, negativity (apex activity). Confusion will be avoided if one always bears in mind that "negativity" (the electrical term) and "activity" (muscular contraction) are synonymous. The Q and S deviations may be exceedingly minute or may be entirely absent. The height of the R wave in any one deviation in terms of R waves in the remaining deviations may be determined by Einthoven's formula, namely R I = R II − R III (Chapter XXXIII). The numerals after the R correspond to the respective leads. The R deviation, the most prominent of all the waves of the normal electrocardiogram, is directed upward a distance varying from 10 to 15 millivolts. As measured at the base line, the time required for its formation varies from 0.02 to 0.05 second. Because of the quick deflection of the string, the sides of the R deviation appear as fine lines, the apex is sharp. The T wave slopes gradually; the down stroke is somewhat thinner than the upstroke, the summit is broad and flattened. The R and T waves are sometimes called the first and second ventricular spikes, respectively. Occasionally a U wave is present. It rises only slightly above the isoelectric line; when present, it is found in mid-diastole. All curves are taken with a known deflection time of the string (Fig. 68). This is determined by rapidly throwing in and cutting out a millivolt of current by means of the rheostat (Fig. 63, P.R) with the string tension at its usual standard (Fig. 68). The limit of accuracy for this deflection time is approximately 0.02 second; that is, the string should cover a distance of one centimeter with a millivolt of current in 0.02 second or less. When slower, the string registers curves inaccurately flattened and low.

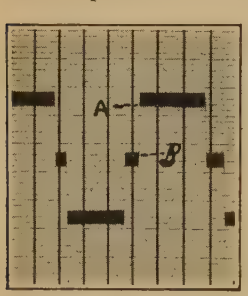


FIG. 68.—Electrocardiogram showing the deflection time of the string. The points A and B are about 0.02 of a second apart.

**Normal Variations from the Standard Electrocardiogram.**—The usual variations are absence of the U wave; marked differences in the height of the R in the several leads; a split R wave with thick sides or summit; abnormally large, flat, or diphasic T waves, especially in leads 2 and 3; a partially inverted T wave in the first lead; a low, flat, or split P wave or its absence in one of the leads; deep Q and S waves, and a so-called QRS complex or as I prefer to designate them from their appearance, M and W complexes (Chapter IX). While the cause of some of these variations is known, the etiology of others is obscure or still in dispute.

The variations of the T wave will be taken up in detail in the succeeding chapter. (Chapter IX.)

**The Normal Electrocardiogram and Its Interpretation.**—A typical standard electrocardiogram—three leads—of an adult with a normal heart is shown in Fig. 69. The R deviation is tallest in the second, the "strong" derivation (Figs. 66, 69), which leads off the current in a direction parallel to the long axis of the heart. It is necessary to describe certain basic facts regarding the electrocardiogram in order that the reader may more readily comprehend the difference in size and direction of the deviations in the various leads in normal and abnormal hearts. As already stated, the electrocardiogram is the graphic representation of the spread of the electrical impulse throughout the heart. With normal cardiac rhythm, the peaks or deviations

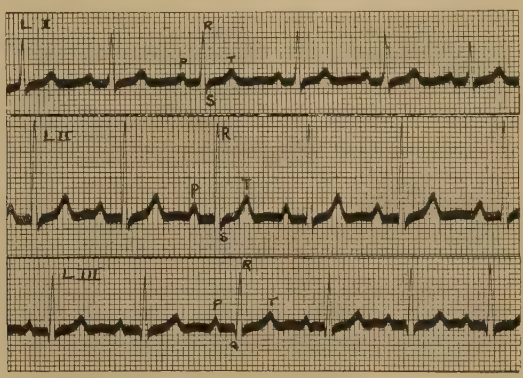


FIG. 69.—Normal electrocardiogram. L I, L II, L III are the three leads. The darker shaded ordinates measure one fifth second; the horizontal spaces, one millivolt each. The S wave is scarcely discernible in L III. The R wave is tallest in L II. (*Courtesy of Dr. A. E. Cohn.*)

of the electrocardiogram represent at any instant of time the sum of total difference of electrical potential in those parts of the heart which are in contraction. The QRS and probably the T deviations are caused by the spread to both ventricles of the excitation wave along the auriculo-ventricular junctional tissue. An upwardly directed R peak denotes contractility of the ventricular base relative to the remainder of the musculature. This may be proved experimentally by placing two electrodes, one upon the right ventricle in front, the other upon the apex; if the right ventricle be first stimulated, the R is deviated upward (base-active); if the apex be first stimulated, then the resultant deviation is downward (apex-active).

**How the "Clinical" Electrocardiogram is Taken** (Fig. 56).—It is best to sit the patient in a room other than that in which the electrocardiograph is placed, in order to obviate the possible effect of nervous excitement upon the heart. In addition, it is always advisable to assure the patient that he will feel no electric shock, and that the entire procedure is absolutely painless. After both forearms and the left leg have been exposed they are rubbed vigorously with a piece of gauze dipped in a strong warm solution of salt water; about ½ cup of salt to a pint of water gives a solution of proper strength.

The exposed limbs are rubbed in order to assure proper electrical contact. The electrodes, the lead foil surfaces of which have been washed with the brine, are then wound and snugly tied about the exposed extremities. The wires, marked R.A., L.A., and L.L. are then placed in the binding posts respectively of the right arm, left arm and left leg. The patient is told to sit or to lay quietly, as the case may be. This finishes the manipulation as far as the patient is concerned.

Attention is now devoted entirely to the electrocardiograph. The room is darkened. Depending upon the source of light, the arc or electric bulb is turned on and focused so as to give a clear image of the projected magnified string. Additional focussing is usually required; this is done by manipulating one or both ends of the microscope (Fig. 63, M) by turning the collar to which the microscope is attached, just as one turns an adjustment screw of the ordinary microscope. The string is now tested. The time marker (Fig. 63 T.M.), which is usually regulated to make and break the light every fifth second, is then set in motion. The result in the electrocardiogram shows vertical parallel lines (Fig. 69), the distance between which represents fifth seconds intervals. There is a small needle galvanometer (potentiometer), arbitrarily set at a certain figure (usually 10°), which is placed in circuit by turning a shunt (Fig. 62): this regulates the voltage. The patient is then placed in circuit by means of a shunt or key shunt (Fig. 63, P.R.). Lead I is the first to be taken. Lead I is marked on the shunt box, that is, the wires that come from right arm and left arm are now in circuit. A millivolt of current is then passed through the string by manipulating the appropriate rheostat, (that is, by turning the handle of that which measures millivolts, the millivolt rheostat). The amount of excursion of the string is then noted either upon a scaled rule measuring centimeters attached to the top of the front face of the camera, or upon a centimeter ruled card toward the bottom of the camera. The card is better because it can be moved so that the image of the string falls upon one of the ruled lines. If it is seen that the string moves less than one centimeter, it denotes that the string is too tight: It is therefore loosened by turning a micrometer screw of the string house (Fig. 63, S.H.) which regulates the tension of the string. Loosening is continued until one millivolt of current makes the string jump exactly one centimeter. If the string jumps more than 1 cm. for 1 millivolt, the micrometer screw is correspondingly tightened. Should the string leave the center of the field during the time the electrocardiogram is taken it can be brought to the center by turning the millivolt rheostat a sufficient number of turns; if that be insufficient, the next larger rheostat (the centivolt bank) can be used. Thus the standard electrocardiogram is taken. During this manipulation the string, being activated by the current from the heart, is of course casting its magnified shadow on the front of the camera box. There are various types of the latter. They all contain a focusing lens with a micrometer lined surface, in order to show in millivolts the height of the

shadow cast by the string (Fig. 63, F.B). They all also contain shutters to expose the film when all is ready, and a motor to turn the film on its spool in the camera box. Plates are sometimes used instead of film. This has the disadvantage that only exposure of a certain length can be employed, while with a film camera the exposure can be made long or short as required, depending upon the type of case.

After lead I is finished all shunts are turned to zero. Then leads II and III are similarly taken; the film is then cut off by a sliding knife and drops in the camera box and is later developed. This is the electrocardiogram. The patient is then disconnected. All these steps naturally seem intricate to the beginner. With practice however, a complete electrocardiogram can be taken in a few minutes after the patient is in circuit.

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## CHAPTER IX

## VARIATIONS IN THE MUSCULAR MASS OF THE HEART THAT INFLUENCE THE RHYTHMIC ELECTROCARDIOGRAM—VENTRICULAR HYPERTROPHY AND DILATATION—SPLIT WAVES—VARIATIONS OF THE T WAVE—SUMMARY OF CLINICAL INTERPRETATIONS.

So much confusion, contradiction and ill-grounded conclusions are found in electrocardiographic literature, especially in the attempt to diagnose hypertrophy and myocarditis from the electrocardiogram, that the physician must continually hark back to fundamental considerations, otherwise electrocardiograms instead of aiding in clarifying clinical interpretation, may have just the reverse effect. There is indeed much unequivocal and definite information that the electrocardiogram can give regarding hypertrophy and myocarditis, particularly in conjunction with clinical data; but I now refer in the main to debatable and questionable interpretations. In that which follows, some of these will be discussed.

From what has been said regarding monophasic and diphasic curves and their fundamental electrophysiological causes (Chapter VIII), it is evident that purely physical considerations affecting a contracting muscle may influence the resultant electrocardiogram. For example, let us in Fig. 60 conceive the muscle to consist of fibers of various lengths running in various directions, instead of being parallel and of the same length; we should then expect some change in the resultant electrocardiogram and not a simple diphasic curve. It has already been pointed out that the muscular architecture of the heart is an extremely intricate one, and that there are layers which run in various directions from one chamber to the other (Chapter I). This phase of pure mass consideration as affecting and influencing the electrocardiogram has been admirably summed up by A. E. Cohn as due to the "disposition and volume of the muscular mass of each pair of cavities." Volumetric and mass considerations apply not only to the involved architecture of the normal heart, but also to diseased and hypertrophied hearts. For example, it seems probable that the hypertrophic process does not always, or even regularly, express itself by hypertrophy of one chamber as compared with the other, but rather as a process affecting the fundamental complicated muscle layers. To such observations should be added the extremely important experimental one that in electrocardiograms of dogs with the chest wall intact, the direction of the main deviation—the R wave—is often changed by turning the animal to one side or the other. This can only mean that such manipulations serve to so change the ventricular axis in

the relative positions of right and left ventricles, that one or the other becomes electrically predominant, and hence correspondingly deviates the R wave. Such experiments have their analogies in the human electrocardiogram, and as will be pointed out later, should serve to caution us in dogmatically diagnosing hypertrophy of a cardiac chamber from the electrocardiogram alone.

Remembering then the purely physical side of the heart as expressed by its mass and volume, and the various shapes and forms the heart muscle mass as a whole may take, it can be understood how changes in muscular volume and mass profoundly influence the size and direction of the electrocardiographic deviations. To use a crass but perhaps expressive comparison, excessive hypertrophy of the left ventricle, by distorting the normal muscular (and therefore the normal electrical) balance of the heart, would have the tendency to "pull down" the excitation wave somewhat as a magnet attracts a needle. It is evident, however, that any classification based upon differences of shape, mass and volume of the cardiac musculature will meet with numerous exceptions, for in the present state of our knowledge of roentgenology and physical diagnosis, it is impossible to diagnose with any degree of exactness, many instances of distorted muscular balance such as are found in hypertrophy and dilatation of the various cardiac cavities. Indeed, it seems conceivable that right and left chamber distortions may balance each other; the resultant effect on the electrocardiogram would therefore be nil. Bearing the above limitations in mind, the following tabulation is offered in an attempt to clarify many of the causes for variations from the normal electrocardiographic standard in the rhythmically beating heart.

**Abnormal Size, Disposition and Volume of the Ventricular Musculature as Affecting the Electrocardiogram.**

A. Horizontally disposed (squatty) hearts.
B. Vertically disposed (drop) hearts.
C. Cardiac displacements.
D. Congenital dextrocardia.
E. Phasic variations with breathing.
F. Ventricular hypertrophy (left and right).
G. Ventricular dilatation (left and right).
H. Abnormal ventricular mobility.

(*A*) **Horizontally Disposed (Squatty) Hearts.**—If from any cause the heart lies abnormally flat on the diaphragm, the ventricles are apt to be "disposed" with a preponderant balance to the left, as diagrammatically illustrated in Fig. 70, D. In some clinical cases of "squatty heart," R III[1] is either dwarfed or negative. Leaving the question of hypertrophy for later consideration, examples of squatty hearts are found oftenest in obese, middle-aged individuals. Electrocardiograms from several such patients are shown (Figs. 71, 72, 73). Cardiac symptoms are rarely present. Fluoroscopic examinations (Chapter XII) reveal the ventricular mass lying flat upon the

[1] The numerals placed after the deviations refer to the latter in their respective leads.

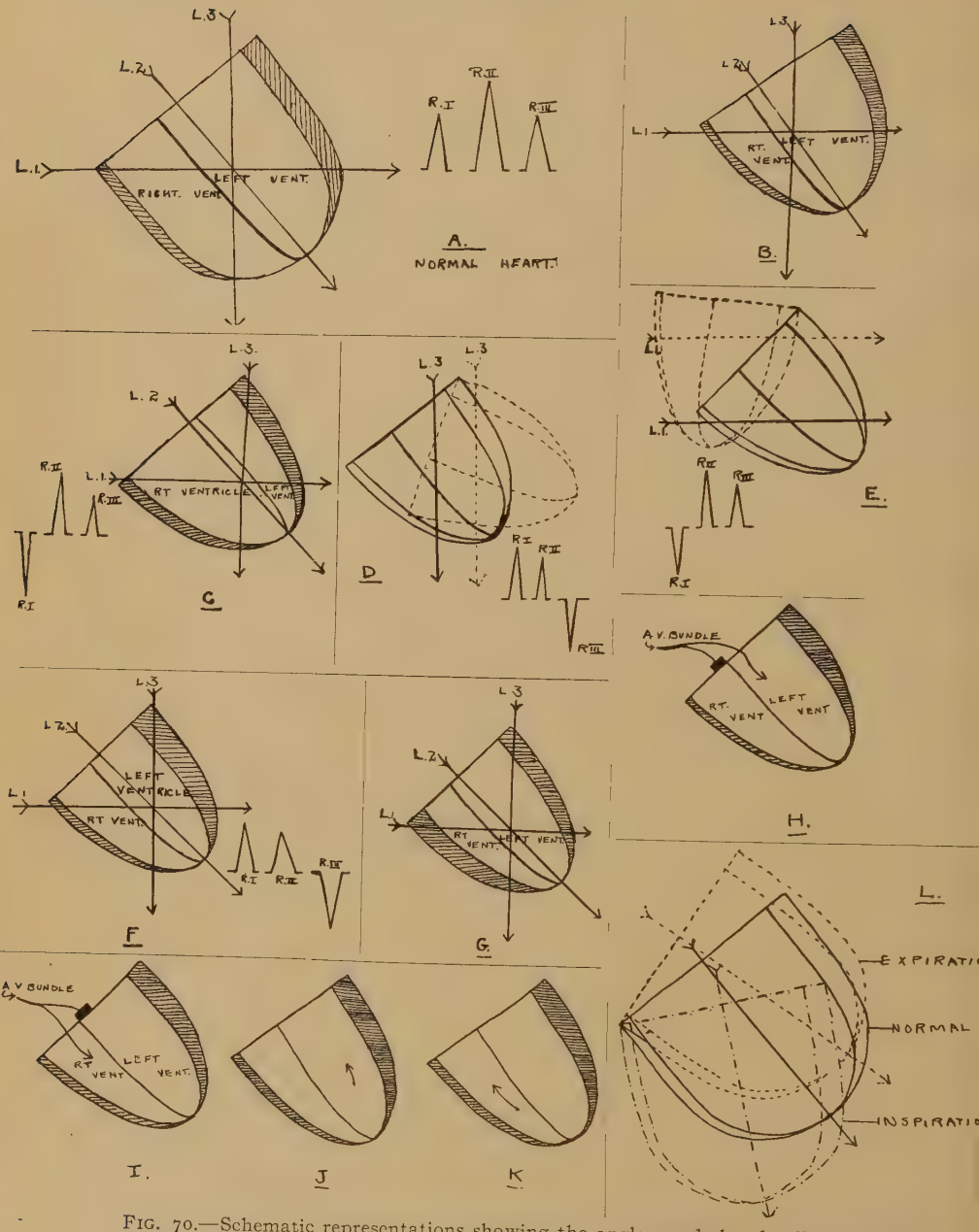


FIG. 70.—Schematic representations showing the angles made by the direction of the leads with varying ventricular axes, and the corresponding electrocardiograms. Although the ventricles are schematically of different sizes, it is here assumed that the volumetric contents of both chambers are alike.

A, Normal heart; B, left ventricular dilatation; C, right ventricular dilatation; D, left ventricular hyperbalance; E, right ventricular hyperbalance; F, left ventricular hypertrophy; G, right ventricular hypertrophy (electrocardiogram as in C); H, block of right bundle branch; I, block of left bundle branch; J, left ventricular extrasystole; K, right ventricular extrasystole; L, change of position of the heart with inspiration and expiration.

diaphragm; the dome of the latter is less curved and the diaphragmatic excursion is reduced in range. These factors are of importance in the etiology of the abnormal position of the ventricles. Gaseous distention of the stomach may have a similar effect upon the position and action of the diaphragm, and hence upon the position of the heart.

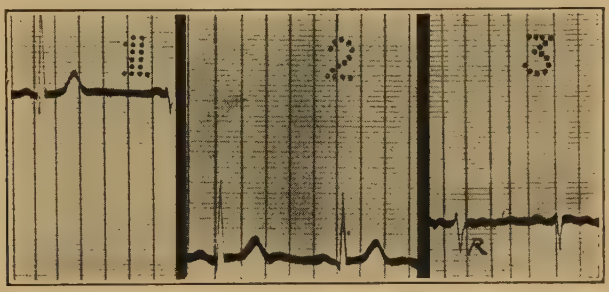


FIG. 71.

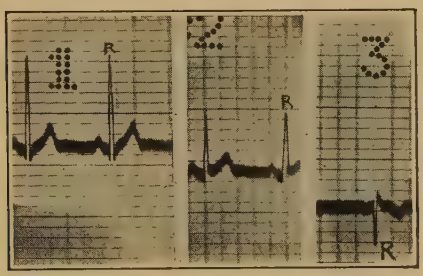


FIG. 72.

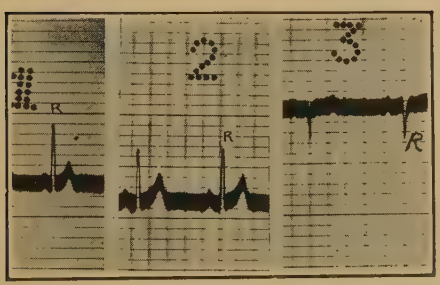


FIG. 73.

FIGS. 71, 72, 73.—Electrocardiograms of patients with normal hearts and with negative R in L III. In all these cases the ventricle lay flat upon the diaphragm.

(*B*) **Vertically Disposed (Drop) Hearts.**—Quite opposite, theoretically, to the foregoing is the distribution of the ventricular musculature when the heart is narrow and lies vertically in the chest. The assumed muscular axis is then diagrammatically represented in Fig. 70 E; R I becomes abnormally small or negative. Clinically, patients with vertically directed (so-called

"drop") hearts are apt to be young, tall and gaunt individuals with loosely hung hearts. With the fluoroscope the entire organ appears narrow and

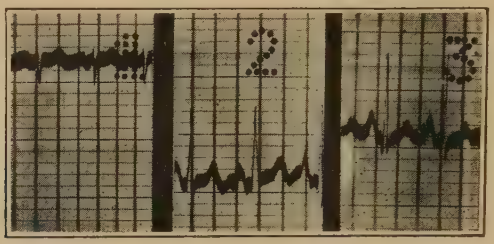

FIG. 74.—Electrocardiogram of patient with narrow heart ("drop" heart) illustrating right ventricular hyperbalance.

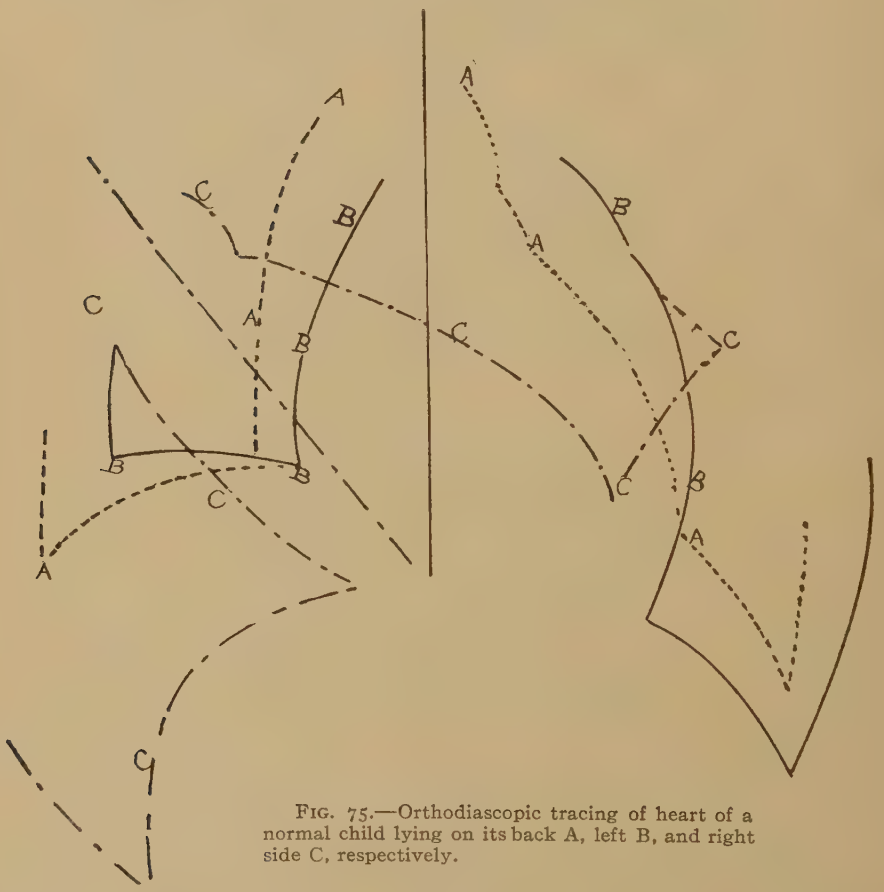


FIG. 75.—Orthodiascopic tracing of heart of a normal child lying on its back A, left B, and right side C, respectively.

graceful, and for the most part hidden behind the sternum. At times, the apex scarcely touches the diaphragm. An illustrative electrocardiogram is shown in Fig. 74.

(*C*) **Cardiac Displacements.**—The heart can be displaced, as is known, by pleural exudates, adhesions, mediastinal tumors, etc. If the heart be displaced laterally so that there is no disturbance of the muscular balance of the heart, the electrocardiogram does not change. The latter is only affected when the new position (made, for example, by tilting the heart) has brought about change in the relationship and disposition of the ventricles, for then the electrical axis as well as the electrical potential of the ventricles is changed. The effect of displacement can be best exemplified in children,

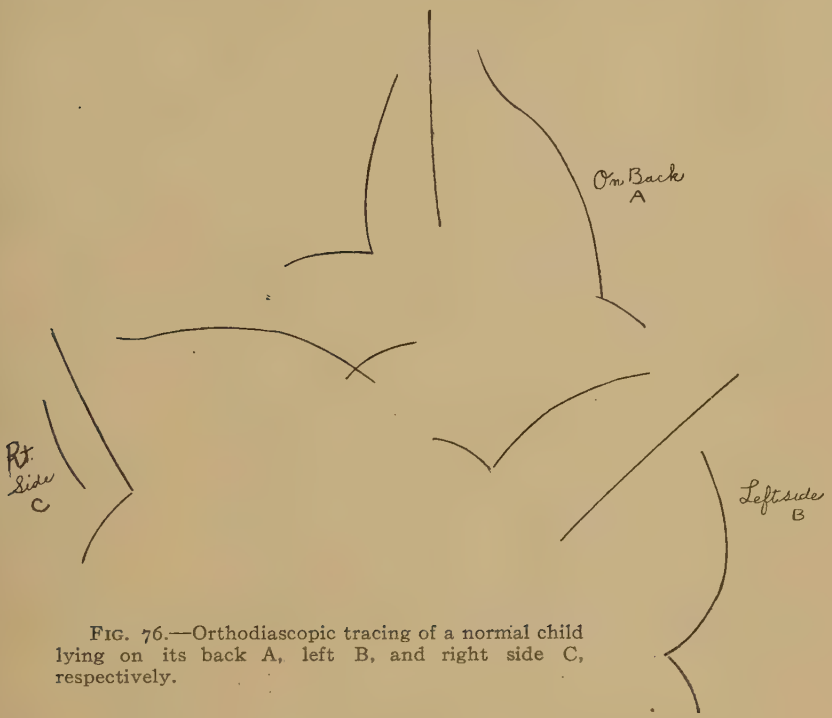


FIG. 76.—Orthodiascopic tracing of a normal child lying on its back A, left B, and right side C, respectively.

and is illustrated in Fig. 76, A, B, C—the orthodiascopic tracings of a healthy boy of ten lying respectively on his back, left and right sides. The tracings show marked variations in the disposition and contour of the heart, mainly due to ventricular rotation and consequent foreshortening or lengthening of the cardiac outline. Another illustration of the mobile heart is Fig. 75 (positions A, B, C), orthodiascopic tracings of the normal heart of a child of seven lying respectively on her back, left and right sides. Corresponding electrograms were taken (Fig. 77, A, B, C). In addition to slight changes in the Q and S deviations, the heights of the R in the A, B, C positions varied as follows: Lead I, R = $6^A$, $5^B$, $6^C$ (the numbers refer to the number of millivolts of deflection); in Lead II, R = $10^A$, $15^B$, $16^C$; in lead III, R = $6^A$, $13^B$, $10^C$.

(*D*) **Congenital Dextrocardia.**—As a corollary to the observations already made regarding the effect of change of the planes of electrical potential upon the electrocardiogram, it is apparent that congenital dextrocardia in the first (sometimes called the symmetrical) lead will produce electrocardiographic deviations exactly opposite in direction to the normal (Fig. 78). All the

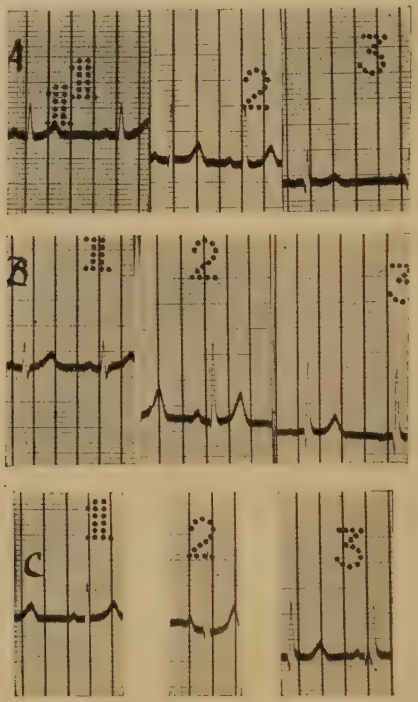


FIG. 77.—Electrocardiogram of a child whose orthodiascopic tracing is Fig. 75. The letters A, B and C again refer to the position of the child on its back, left and right side respectively.

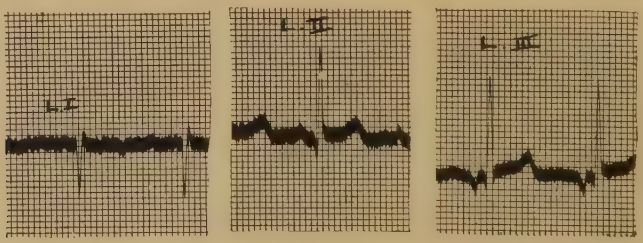

FIG. 78.—Congenital dextrocardia. All the deviations are directed downward in L I. R III is taller than R II.

peaks are directed downwards instead of upwards; R III becomes taller than RII. Such an electrocardiogram offers indubitable proof of congenital dextrocardia and serves to distinguish the latter from acquired rightsided

malposition of the heart due to fluid in the chest, pulmonary tumors, pneumothorax, adhesions, etc. For example, Fig. 79 is the electrocardiogram of a patient with an acquired dextrocardia, in whom the heart was drawn into the right chest by adhesions following a right-sided pulmonary abscess and subsequent pleural fistula from thorocatomy. Fluoroscopically, the heart was seen to occupy an area in the right chest practically identical with that of congenital dextrocardia. Since the heart had been pulled in a *lateral* direction only, the electrocardiographic deviations remained normal in direction.

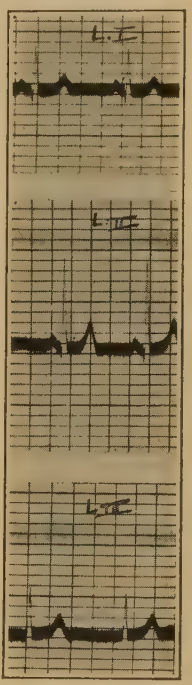

FIG. 79.—Acquired dextrocardia with normally directed deviations in all three leads.

(*E*) **Phasic Variations with Breathing.**—As the result of breathing, some electrocardiograms, usually in the second and third leads, present a rhythmic waxing and waning, an increase and decrease respectively in the size of the R waves. In fluoroscopy, I have often noted a marked variation in the position of the heart during respiration (Fig. 70, L,) sometimes sufficiently pronounced to produce a movement of the apical portion of the ventricle through an arc of several centimeters. This shift is most evident in younger individuals with thin chest walls and large respiratory excursions of the diaphragm; it is least noticeable in patients with fat abdominal walls and broad hearts. The heart moves with its base as a comparatively fixed point. During inspiration there is a descent in a clockwise direction of the ventricular mass, especially of its apical portion; the heart tends to assume an erect posture. The rise of the diaphragm during expiration produces a contrary effect, the left ventricle then moving anti-clockwise. These movements, when marked, necessarily affect the muscular relationship of the ventricles and, as a consequence, alter the electrocardiogram. This is usually most noticeable in the third lead, probably because the left ventricle is especially influenced by the respiratory phases. R III becomes taller during inspiration and smaller during expiration as the ventricular mass tends to assume a more vertical or horizontal position, respectively (Fig. 70, L).

Respiratory phasic variations are also found in patients with ventricular hypertrophy, but since the cardiac excursion is ordinarily less, the phasic electrocardiographic variations become correspondingly limited. Figures 80 and 81 are examples of a moderate respiratory effect upon the size of the deviations. Figure 81 is the electrocardiogram of a stout individual with a normal heart lying in a squatty position; Fig. 82, of a patient with left ventricular hypertrophy. When R III is small, phasic variations may not only dwarf it but may cause its final disappearance, as shown in Fig. 82.

(*F*) **Ventricular Hypertrophy—Left and Right** (Fig. 70, F.G.).—It has sometimes been assumed that negative R I (S I) and positive R III are indicative of right ventricular hypertrophy, and positive R I and negative R III (S III), of left ventricular hypertrophy. This observation has been partly substantiated by necropsies. On the other hand, exceptions have been noted both at postmortem and as the result of clinical observation. Some undoubted cases of left ventricular hypertrophy, for example, do not give the deviations assumed for this condition. Besides, it is by no means infre-

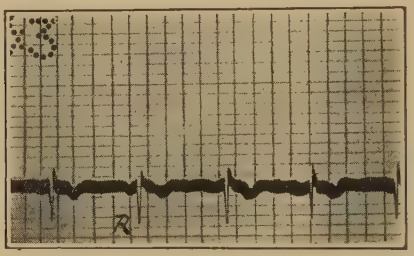

FIG. 80.—Slight phasic variations with breathing.

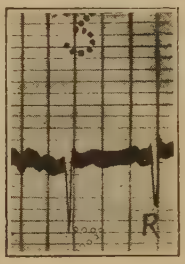

FIG. 81.—L II. Slight phasic variation with breathing.

quent to find perfectly normal hearts in a normal position in the chest, which present the deviations supposedly typical of hypertrophy. It will thus be seen that there are many drawbacks and exceptions to definite conclusions regarding the existence of hypertrophy based upon the direction of the electrocardiographic deviations alone. Again, a case may show undoubted clinical evidence of left ventricular hypertrophy, the X-ray may show the heart in a normal position as far as the cardiac axis is concerned; and yet a deep negative deviation in the third lead (S 3) may be missing. In

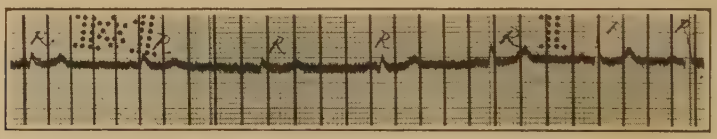

FIG. 82.—Marked phasic variation with breathing.

such instances one can only assume that there is a right as well as a left ventricular hypertrophy, and that therefore the muscular (and with it the electrical) balance has not been disturbed sufficiently to distort or "pull down" the third deviation and make it negative. In other words, with a heart that shows definite clinical evidence of left ventricular hypertrophy, whose axial position in the chest is normal, and whose electrocardiogram shows spikes normally directed, it is fair to assume that the electrocardiographic preponderance due to left ventricular hypertrophy has been offset by a right ventricular one.

It has already been shown that a squatty (transverse) and a vertical (elongated) heart may yield abnormally directed deviations in the third and first leads respectively, even though these organs are otherwise normal. Applying these considerations, for example, to a heart that not only lies too transversely but also has a hypertrophic left ventricle, it would mean that the third lead would be disproportionately negative for that amount of hypertrophy; that is to say, there would be a summation and re-inforced negative deviation due both to the abnormally flat position of the heart and to the hypertrophy. If, on the other hand, a left-chambered hypertrophic heart were too vertical in the chest, it would tend to decrease the length of the negative deviation in the third lead. Finally, it seems probable that the influence of ventricular dilatation (see Fig. 70, G) may possibly counterbalance or at least vitiate in one direction or another electrocardiographic deviations due to hypertrophy alone. All these factors necessarily influence electrocardiographic inferences regarding hypertrophy. It is evident also that these factors themselves or their interdependence cannot always be known; hence, electrocardiographic formulae which deal with electrocardiograms alone and which give no information regarding the direction of the cardiac axis in the living, or the relative hypertrophy and dilatation of the ventricular chambers at autopsy omit important data for proper inference. Even with the data regarding the direction of the cardiac axis in the living, it seems a difficult mathematical as well as clinical problem to determine the correlation and relative importance of the known and unknown quantities. Hence electrocardiographic formulae for hypertrophy, unless carefully construed from various aspects, may cause misleading information. On the whole it is better to speak of the electrocardiogram as showing ventricular predominance or preponderance than ventricular hypertrophy, for, as just indicated, other considerations must be weighed before the diagnosis of hypertrophy can be concluded from the electrocardiogram alone. As a general rule, however, it will be found that when the R I is tall and positive, and R III deep and negative, left ventricular hypertrophy exists. This is clinically best exemplified in aortic disease with left ventricular hypertrophy. Examples are given in Figs. 83–85. Occasionally, however, R III is positive, even when left ventricular hypertrophy is present (Fig. 86).

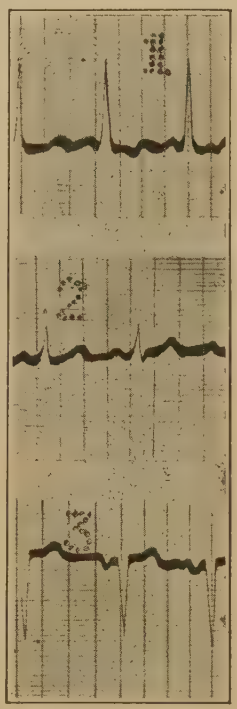


FIG. 83.—Aneurismal dilatation of the aorta with left ventricular hypertrophy. Negative deviation in Lead III.

Naturally, as in the left, similar considerations govern electrocardiographic conclusions regarding right ventricular hypertrophy. The electrocardiographic evidence of right ventricular hypertrophy is best exemplified by congenital cardiac disease due to malformation of the pulmonary artery. This is illustrated in the case of a boy of 17 with patent ductus arteriosus

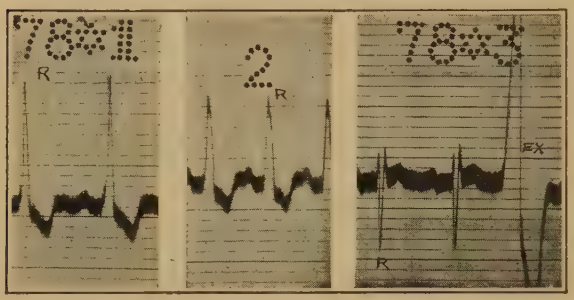


FIG. 84.—Aortic stenosis. Left ventricular hypertrophy. Negative R in L III. Ventricular extrasystole (Ex) in ead III.

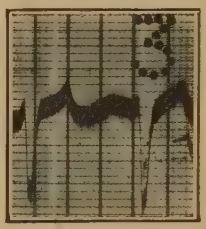

FIG. 85.—Negative ventricular deviation in lead III. From a case of left ventricular hypertrophy and aortic aneurism.

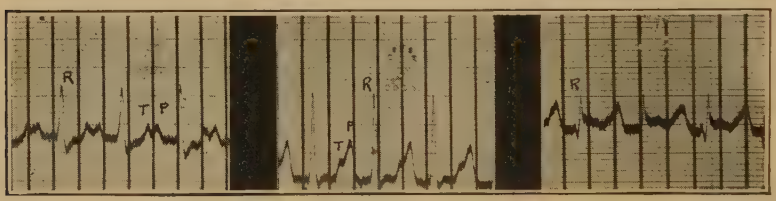


FIG. 86.—Electrocardiogram of patient with aortic regurgitation and left ventricular hypertrophy; the R deviation is positive in L III.

(Fig. 87). Both R I and R II are negative. Upon the theory of mass imbalance affecting the electrocardiogram, it would seem that a negative R II in addition to negative R I is indicative of very marked right ventricu-

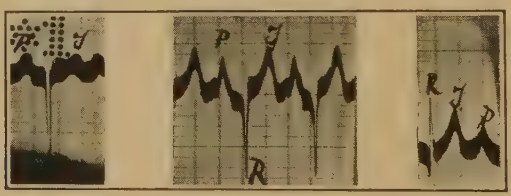


FIG. 87.—Electrocardiogram of a boy of 17 with congenital patent ductus arteriosus Note negative R I, R II. evidence of marked right ventricular hypertrophy.

lar hypertrophy. The same theoretical consideration applies to negative R II and R III as indicating extreme left ventricular hypertrophy. This is exemplified in Fig. 88 taken from a patient with aortitis, and with clinical and fluoroscopic evidence of extreme left ventricular hypertrophy. Infants

and young children are also apt to have the electrocardiographic complex of right ventricular enlargement, because in them the walls of the right ventricle are relatively thick.

Cases of mitral stenosis do not always yield electrocardiograms indicative of right ventricular hypertrophy, a condition usually associated with this

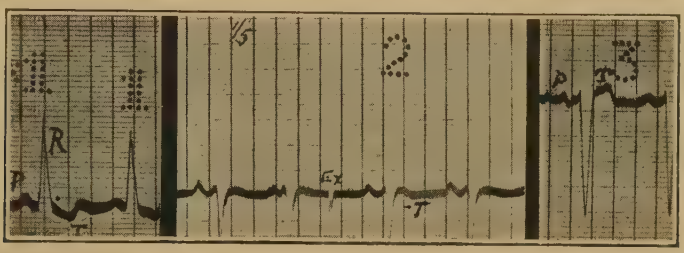


FIG. 88.—Electrocardiogram showing an interpolated extrasystole (L III, Ex). R is abnormally wide, especially in L I and L III. The direction of the R deviations in the various leads indicates marked left ventricular hypertrophy. T I and T II are negative, presumed evidence of myocarditis. (From a patient with left ventricular hypertrophy and luetic aneurismal dilatation of the entire thoracic aorta.)

lesion. Thus, Figs. 89 and 90 are from cases of marked typical stenoses. In Fig. 89, the main ventricular deviation is negative; in Fig. 90, it is positive. Whether similar electrocardiograms in cases in which right ventricular hypertrophy is clinically assumed, are due to some of the factors already discussed (abnormally disposed ventricles, ventricular dilatations or hypertrophy) or whether they may even be due to counterbalancing left ventricular hypertrophy, it is at present impossible to state.

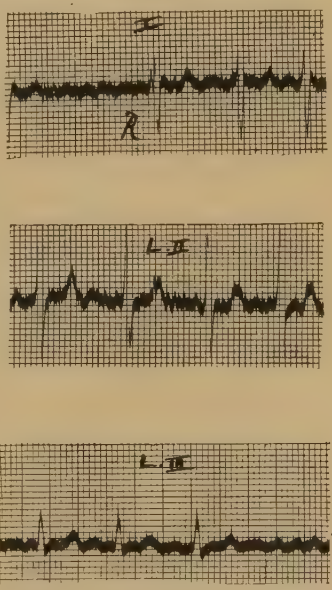


FIG. 89.—Advanced mitral stenosis. R I is negative. Auricular fibrillation is present.

(*G*) **Ventricular Dilatation** (Fig. 70, B.C.).—Although hearts which are organically sound may occasionally become temporarily dilated as the result of overstrain or of tachycardial attacks, the term ventricular dilatation is here meant to apply to decompensated, diseased hearts. To a great extent, our clinical knowledge regarding cardiac dilatation, especially of one chamber as contrasted with the other, or of dilatations as differentiated from moderate hypertrophy, is meager (Chapter VI), although autopsy findings of dilated cavities are by no means infrequent. There exists, however, some definite evidence that cardiac dilatation following acute endocarditis is not uncommon in children. This subject has never been

studied from the electrocardiographic aspect, but, as with considerations regarding the effect of mass changes, it seems probable that muscular redistribution resulting from dilatation can also bring about changes in the electrocardiographic deviations.

I possess some electrocardiographic data which, when combined with clinical examination, have a bearing upon my assumption that changes in

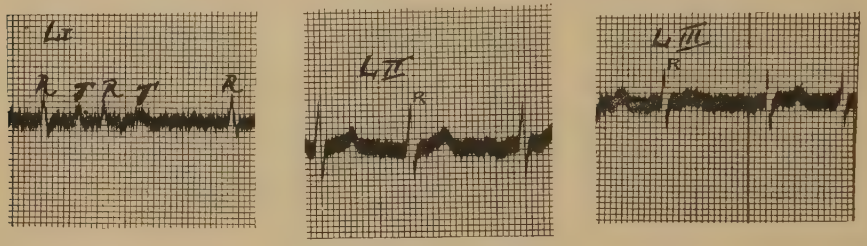

FIG. 90.—Advanced mitral stenosis with auricular fibrillation. All the R deviations are positive.

the volume of ventricular chambers from dilatation and consequent distortion of ventricular mass relationship, can bring about changes in the direction of the ventricular spikes of the electrocardiogram. Thus in a few instances of patients with severely decompensated valvular lesions, there was a definite change in the direction of the ventricular deviations in at least one lead shortly before death. This change was not due to any bundle lesion (Chapter X), for the electrocardiograms presented no such evidence. Naturally sudden hypertrophy as a terminal condition could also be ruled out. In my opinion, such an electrocardiographic change from positive to negative or the reverse could be brought about by but two causes: Some force from without (*e.g.*, pericarditis with effusion) causing sudden compression of one ventricle and thus disturbing the pre-existing interventricular mass relationship; or changes in the degree of dilatation of the ventricular chambers themselves. The latter appears the more plausible since the changes occurred in severely decompensated hearts shortly before death. Autopsies were not obtained.

**Abnormal Ventricular Mobility (H)—Split and Slurred R Complexes—M and W Wave—Q R S Complexes.**—Split, slurred and various types of abnormal ventricular waves have been described. About them much confusion has arisen as to their etiology, and especially as to their clinical significance. I shall attempt to group and separate these abnormal waves into somewhat more orderly groups, and as far as possible shall give my interpretation of them from the clinical and experimental standpoints. In this manner, even if the etiology be still partially or entirely unknown, we shall at least be able to state with fair accuracy that some of these bizarre complexes, while of extreme interest from the electro-physical and electrocardiographic standpoints, do not of themselves indicate cardiac disease in man.

I shall first take up those split complexes which I have termed M and W deviations (Figs. 93, 94, 95) because they so closely resemble these letters.

During the course of routine fluoroscopic examination of normal and abnormal hearts, I have encountered occasional cases in which there was a sort of to-and fro tilting, rocking motion of the ventricular mass, with the base of the heart acting as the fulcrum. At times the entire ventricular mass, at others only its apical portion was seemingly involved in this abnormal motion. Before I had the opportunity to attempt experimental splitting of the R wave in electrocardiograms of narcotized dogs, my conception had been that this abnormal rocking or tilting motion of the ventricular mass interfered with the mass relationship of the ventricular chambers, and thus brought about a condition in which the ventricular excitation wave was really repeated; in other words, a split M or W wave really consists of two component R (M) or S (W) deviations, respectively. I would emphasize that I refer only to split waves whose measurement at the base line shows that they have not taken an abnormally long time for their production: That is, the entire width of the wave is not longer than 0.07 of a second. Although I believe clinically that abnormal rocking or tilting of the heart during ventricular contraction has a decided influence in causing these split waves, experimental observation[1] however has caused me to partially change my views as to their etiology. The essence of these experiments has been as follows: If the forelimbs and left leg of a narcotized dog be connected with the electrocardiographic apparatus, the chest opened, a hook be introduced in either

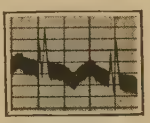

FIG. 93. — Electrocardiogram showing split R (M) complex.

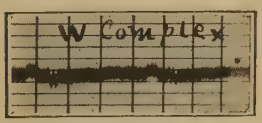


FIG. 94.

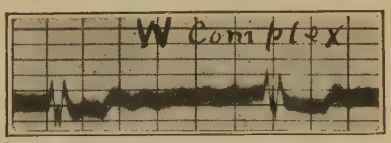


FIG. 95.

FIGS. 94, 95.—Electrocardiograms showing split R (W) complexes.

ventricle, and the ventricle be then slowly or violently twisted in any direction, it will not cause splitting or even slurring or thickening of the main ventricular deviation (R or S wave). There are however two methods by which splitting (M or W waves) can be produced. One is by introducing a slightly bent, long steel sound through the jugular, superior vena cava and tricuspid orifice so that the sound rests against the inner ventricular surface of the right ventricle. Tilting of the exposed end of the sound in the neck in such manner that its ventricular end is held firmly against the

[1] These experiments, as yet unpublished and incomplete were done in conjunction with and through the kindness of Dr. Edwards, of the Department of Physiology, Cornell University.

ventricular wall will in the majority of instances cause M or W complexes. This of course has no analogy in man. It demonstrates however that any physical influence which can tilt out even slightly, the right ventricular wall (it is experimentally difficult to introduce a sound in the left ventricle) may split the main ventricular complex into two component parts. The second experimental procedure consisted in pressing two fingers through an abdominal incision against the under surface of the diaphragm. The fingers were pressed against that part of the heart—right or left ventricle—which beat most forcibly. As a result, the R or S waves were also split into M or W complexes, respectively.

While the underlying electro-physiological cause of these experimentally split complexes has not as yet been ascertained, what happened to the ventricles was apparently some physical disturbance of the ventricular balance. With the sound introduced into the right ventricle, there was presumably slight ballooning of a small ventricular area: With the fingers pressed against the under surface of the diaphragm, the heart bobbed or pounded against the fingers, thus producing a sort of muscular rebound. Perhaps some similar physical ventricular disturbance happens clinically when the healthy or the diseased heart overacts or tilts during ventricular contraction. At any rate, the experiments show that purely physical facts can account for the M and W split waves. Hence alone they cannot be used as evidence of myocardial disease.

In addition to splitting of the R wave from causes just described (variations due to change in relative position and volume of the ventricular musculature), asynchronous activity of the chambers may also possibly act in a similar manner. There are conditions in which, for various reasons, there is

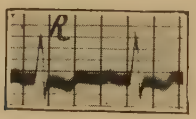


FIG. 91.—Electrocardiogram showing somewhat thickened R summit.

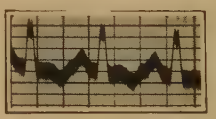

FIG. 92.—Electrocardiogram (L II) showing slightly notched R summit.

a retardation of the excitation wave in one chamber as compared with its fellow. Such retardation may thus also account for many of the "split" and "notched" complexes. The normal difference in contraction time between the two ventricles may amount to as much as 0.03 second. If there be but slight retardation of the excitation waves in the ventricles due to abnormal ventricular asynchronism, it may account for the thickening of the R deviation at its summit (Fig. 91) as well as for various degrees of notching at the apex (Fig. 92).

Similar in etiological significance to the notched R waves from asynchronous ventricular activity are the notched or split P deviations (Fig. 96); these occasionally give rise to the appearance of two distinct undulations.

Such complexes are probably due to asynchronous auricular contractions. A notched P is found most often in mitral stenosis.

**Intraventricular or Arborization Block.**—A QRS complex has been described (Oppenheimer and Rothchild) in which the time required for the

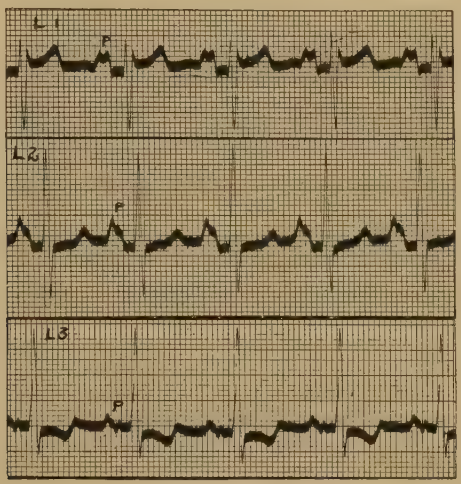


FIG. 96.—Notched P wave from a case of mitral stenosis. (*Courtesy of A. E. Cohn.*)

completion of this group was prolonged beyond 0.10 second, the extreme normal limit. The other characteristics were an R wave abnormally broad; instead of clean and sharp sides, it was slightly or considerably notched and

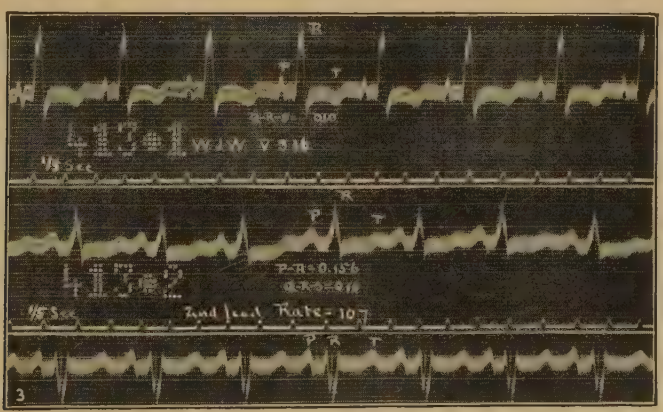


FIG. 97.—Electrocardiogram showing intraventricular block. (*Courtesy of G. C. Robinson.*) Note the notched and splintered complexes.

broken. The waves were usually of low amplitude in all leads. This bizarre electrocardiographic complex was found in cases in which there was marked arteriosclerotic or cardio-renal disease, usually associated with severe myocarditis. In four cases, the clinical picture was corroborated by necropsy

examination; this showed sclerosis especially in the endocardial and subendocardial layers; that is, in the neighborhood of the terminal arborizations of the conduction system. This abnormal electrocardiogram was considered to be the result of intraventricular arborization block, an interference with the normal and orderly spread of the electrical excitation wave throughout the heart. It should be pointed out, however, that hearts presenting the above clinical or pathological picture need not necessarily produce the abnormal electrocardiogram described. For example one of my

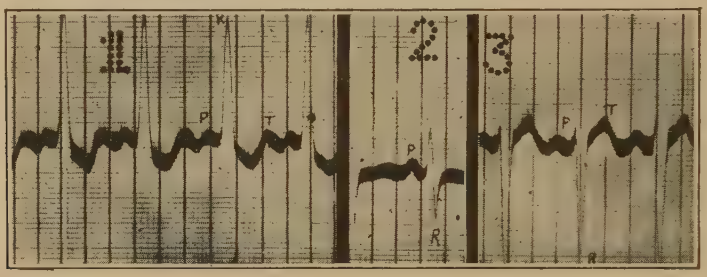


FIG. 98.—Electrocardiogram showing ventricular complexes of abnormal width but of normal form. R is not abnormally wide in all leads.

own cases of the above type of cardio-sclerosis that came to autopsy presented a normal electrocardiogram during life. Other exceptions have also been reported. Until the causes for such *negative* findings in the electrocardiogram have been established, the diagnostic value of this type of electrocardiogram must necessarily be limited. Another type of case has been reported (G. S. Robinson) whose QRS complexes also required 0.10 seconds or longer for their completion (Fig. 97). These were assumed to be due to derangement of intraventricular conduction from hindrance either in the rate or in the path followed by the excitation wave. This hindrance was regarded as due to functional fatigue. In most of the cases from whom the electrocardiograms were taken there was clinical evidence of a profound disturbance in the muscular efficiency of the heart.

**The "Wide R"** (For Detailed Study, See Chapter XXXII).—Both of the above types of intraventricular block are characterized, as has been stated, by a broken and notched complex comprising the QRS group. In an entirely different category belong those cases that I have studied, in whom the R deviations are normal in shape and form, whose sides are unbroken and unnotched, and in whom there was no evidence of a bundle branch lesion. The characteristic of my cases was the abnormal length of time required for the completion of the main ventricular wave. The normal time for the completion of the latter varies from 0.02 to 0.05 second. I have adopted as a standard, a width of 0.07 or over as being an "abnormally wide R." A few cases with their electrocardiograms are herewith epitomized: Fig. 88, is from a patient who had general anasarca from luetic cardiosclerosis. R I=

0.13 of a second in width. Figure 98 is from a case of dilatation of the arch of the aorta and moderate left ventricular hypertrophy; the clinical diagnosis was cardiosclerosis. R I = 0.12, R III = 0.08. Figure 99 is taken from a physician of 53 who developed scarletinal nephritis at the age of 15. At the

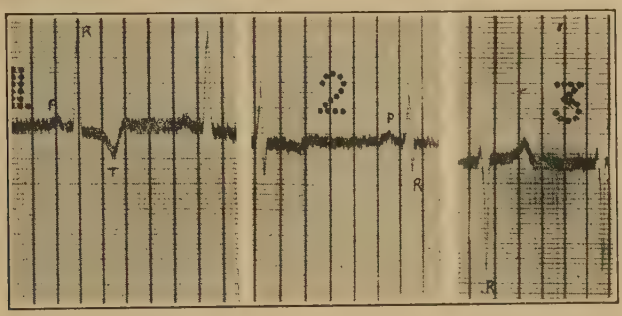


FIG. 99.

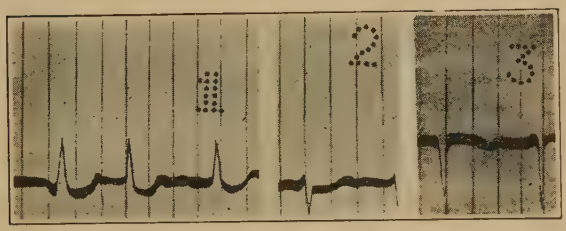

FIG. 100.

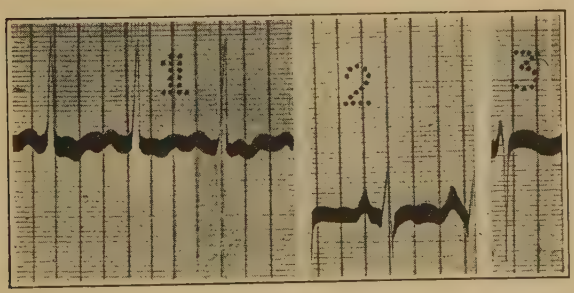

FIG. 101.

FIGS. 99, 100, 101.—Electrocardiograms showing ventricular complexes of abnormal width but of normal form. R is not abnormally wide in all leads.

time of examination the urine contained albumin and casts. The patient had the physical signs and symptoms of moderate cardio-nephritis. R I and R II each equals 0.07. Figure 100 is that of a woman of 68 who, when first seen, was suffering from anasarca as the result of cardio-renal disease. R I = 0.09. Figure 101 was from a male patient of 55 suffering from hypertension

and moderate cardiosclerosis. R I = 0.07. The electrocardiograms of all these patients were taken during the stage of compensation.

In all, I have studied eighteen cases who presented an R width of 0.07 of a second or over. All of these showed unmistakable signs of severe cardiac disease, and at one time or another were decompensated. Clinically, most of the cases had hypertension and left ventricular hypertrophy. Myocardial insufficiency alone was not the cause of the abnormally wide complex, for most of the electrocardiograms were taken when symptoms of decompensation were slight or absent. Ventricular dilatation, another possible assumption for the abnormally wide R, can probably be disregarded for the same reason. Besides, in a series of decompensated valvular cases which I observed in whom ventricular dilatation was presumably a marked feature, the R complexes were of normal width.

Although the fundamental cause of the wide R in my cases is not apparent, it is probably due either to delay in the development of electrical excitation or to delay in its propagation. Since the complexes are normal in form, the excitation wave has presumably followed a normal path in the ventricles. Delay in propagation seems the more probable factor. As in intraventricular block in which severe cardiosclerotic disease is assumed to be the cause of the notched and delayed QRS complex, a wide R of normal shape but of abnormal width may conceivably be due to patches of myocardial thickening scattered throughout the ventricular wall in amounts sufficient to impede and abnormally prolong the excitation wave.

It should be mentioned that I have studied the complexes of other cardiovascular cases who were clinically as ill as the "wide R" patients but who presented no abnormally broad complexes. The reason for this I have not been able to discover.

Of the eighteen cases studied, the R complex was rarely abnormally broad in all leads. Since the first, second, and third leads "draw off" the cardiac current (Chapter VIII), breadthwise, diagonally, and lengthwise, respectively, one may possibly assume that the diseased myocardium lay chiefly in one cardiac plane, thus producing a wide R in the corresponding lead. In a general way, those cases which clinically showed the most myocardial disease were the ones in whom the R complex was widest.

**The T Wave.**—The T deviation is normally monophasic and directed positively (*i.e.* upward) in the three leads (Figs. 67, 69), its height varying between two and three millivolts. It is occasionally quite tall (Fig. 102), sometimes exceeding in height the corresponding ventricular spike (Fig. 103). Not infrequently it is deviated downward in the third lead (Fig. 104) in individuals with normal hearts.

As will be described later (Chapter XXXII) a positively deviated T denotes that the excitation wave has entered the ventricular base. In a number of cases of organic heart disease, especially of the myocardium, T I, T II, or both may be small or negative, (Figs. 84, 88). Such T waves when

negative, indicate that the excitation wave has terminated toward the apex of the ventricles instead of toward the base, the normal termination. Much diagnostic clinical significance has been given to these negative T deviations in the first and second leads, especially since Einthoven regarded a well-

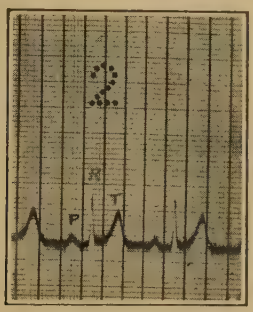


FIG. 102.—Electrocardiogram showing tall T deviation.

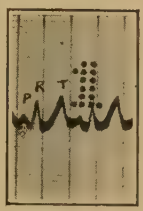


FIG. 103.—Electrocardiogram showing a T deviation taller than R.

marked positive T as a sign of good cardiac contractility, and its absence or diminution, the reverse. It is, however, now known that the T wave may be absent in normal, and be very well marked and positive in diseased hearts. And since the clinical conditions in which the T wave is diphasic or deviated downwards in the first and second leads are only imperfectly understood, clinical inference drawn therefrom can for the present be only tentative.

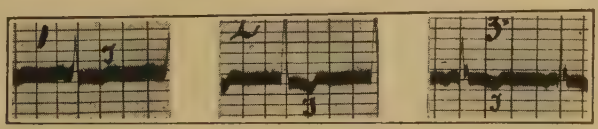

FIG. 104.—Electrocardiogram showing a negative T deviation in leads II and III in an individual with a normal heart.

Experimental investigation has shown that ligation of any large branch of the left coronary artery in dogs is followed by a fairly constant change in the T wave: In the majority of experiments, the direction of the wave was changed from strongly positive to strongly negative in all leads. The negative T remained several days and gradually reverted to the normal form. Clinical corroboration of these experiments (a few cases of occlusion of a branch of the coronary artery) have been reported and confirmed by autopsy in which the T deviations were negative in two of the leads. One of these negative T waves was especially deep.

**Effect of Digitalis on the T Wave.**—It has been demonstrated that digitalization of a patient with heart disease may convert a positive T to a flattened or negative wave. Although this may be partly due to muscular ventricular redistribution as the result of relief of decompensation, experimental evidence indicates that the chief factor is probably due to alteration in the contractil-

ity of the heart muscle. Pharmacological experiments in frogs and mammals have shown that digitalis produces diminished diastolic relaxation of the apical region. In the toxic stage there is systolic standstill of the apex, while the base relaxes in diastole. It is possible, therefore, that similar influences affect the digitalized human heart, producing continued apical activity and thus a negatively deviated T wave. The usual effect of digitalis administration on the T wave may not become evident until atropin has been injected. Atropin apparently unmasks and removes the inhibitory effect of the drug; thereafter the full influence of digitalis on the heart can be observed. Further observation of the clinical effect of digitalis on the T wave is reserved for the study of the dosage and use of that drug (Chapter XX).

Some years ago, Einthoven made the observation in one case that as the result of exercise, a negatively deviated T III in a normal heart became positive. In one instance, I made the reverse observation, namely, a normal positively directed T III became negative (Fig. 105) when the patient was made to breathe sufficiently rapidly to race the heart somewhat. How long T III remained negative I do not know. The patient, a boy of 18, had no organic disease. He had been suffering from tachycardia and from cold

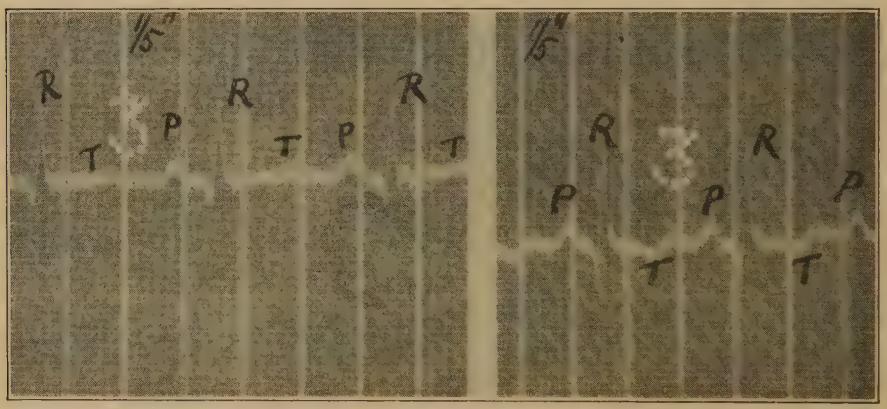


FIG. 105.—Electrocardiogram illustrating the production of a negative T in lead III as the result of rapid breathing. A. quiet breathing; B, forced breathing.

and bluish hands for years. The thyroid was normal. The diagnosis was functional tachycardia and vaso-motor disturbance, a combination common in those with "irritable hearts." I shall later point out (Chapter XVIII) that this symptom complex is probably the result of hyperexcitability of the sympathetic nervous system. Perhaps, then, racing a heart already under the influence of the cardiac accelerators (the sympathetic nervous system) can have a different effect upon the T wave than when such an abnormal accelerator influence is absent.

It has also been recently demonstrated in a few cases that a negative T deviation in decompensated hearts may become positive with restoration

of compensation. The change is probably the result of nerve influences occurring during exercise. This view is based upon the fact that exercise is accompanied by accelerator excitation, and that various experimental observations have shown definite correlation between accelerator stimulation and the size of the T wave. It therefore seems probable that a positive T produced by exercise, or by the restoration of compensation, is due to a neurogenic influence acting upon the ventricular base. Whether, under these conditions, a positive T is to be regarded as a favorable change is an open question and awaits further corroboration; hence clinical inference respecting its value should for the present be guarded.

It is pertinent to inquire why the T wave, the final end of the excitation process in the ventricles, terminates in the ventricular base. It is known physiologically that in all muscular contractions there exists an end deflection of the electrocardiogram following actual muscular contractions, although the exact path followed by the excitation process may not be known. Perhaps in the human heart the terminal waning of the excitation process may be under a nerve influence that shortens or prolongs the excitation process in some parts of the heart more than others, and hence in abnormal instances it may serve to bring about a change in direction of the final deflection, the T wave. (Lewis.)

**Summary of Permissible Clinical Interpretations of Myocarditis and Hypertrophy from the Electrocardiogram Showing Normal Rhythm.**—I shall not here refer to arrhythmias, themselves often used as criteria of organic disease, nor to bundle branch lesions; both are described elsewhere (Chapters X, XI).

Having given the reasons and sounded the warnings regarding what I deem equivocal and questional interpretations from abnormal and bizarre looking electrocardiograms, I shall briefly summarize the positive evidence within our present knowledge.

An abnormally wide, non-notched, non-split R or S in one or more leads, that requires more than 0.07 seconds for its completion, is probably indicative of primary or secondary myocardial disease.

A split QRS (M or W complex) that is completed within a normal time (0.07 of a second or less) is evidence neither for nor against myocarditis. It may be found both in healthy and in diseased hearts.

A notched, splintered or low QRS complex that requires an abnormal time (more than 0.10 of a second) for its completion is evidence either of a special type of myocardial disease (arborization block) or of myocardial fatigue; in either case myocarditis is present.

A negative T in two of the leads, especially if one of the waves be very deep and sharply pointed, is evidence of myocarditis, probably from occlusion of one of the smaller coronaries. Conclusions regarding myocarditis drawn from negative T waves are of no value when the patient has been digitalized.

The presence alone of any of the above abnormal or bizarre electrocardiographic complexes is of diagnostic value. Their absence does not preclude organic heart disease: Indeed, severe heart disease of all types and degrees may be present and yet the electrocardiogram may be practically normal in appearance.

The presence of an abnormal or bizzare electrocardiogram is rarely a clinical measure of the degree of myocardial disease or efficiency. The reason for this probably depends upon the fact that abnormal complexes depend upon the location, rather than upon the amount of myocardial damage.

Mere thickening or slurring (without actual splitting or division) of the R or S wave is found both in healthy and diseased hearts. Hence, alone it does not indicate organic heart disease.

The clinical diagnosis of left ventricular hypertrophy (as against electrocardiographic evidence of preponderance) is permissible when R I is tall. S II moderately deep and S III very deep; of right ventricular hypertrophy (as against electrocardiographic preponderance) when S I is deep, R II moderately tall, and R III quite tall. Ventricular hypertrophy may be present clinically, yet the deviations be normal in direction. Small abnormally directed deviations are of no clinical value in the diagnosis of ventricular hypertrophy of either chamber, because so many other causes may produce these abnormally directed deviations.

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## CHAPTER X

# THE ARRHYTHMIAS—THEIR POLYGRAPHIC, ELECTROCARDIOGRAPHIC AND CLINICAL RECOGNITION

In this chapter are grouped pulse and cardiac irregularities illustrated either by electrocardiograms or polygrams, or both. The object of this apparently promiscuous use of both methods of registration is to accustom the reader to think of the curves in terms of auricular and ventricular activity, no matter what form of graphic representation is used. Reduced to their simplest elements, all types of arrhythmias, even the most complex, depend essentially upon abnormal activity of one or both cardiac chambers. It therefore seems best to accustom the mind and eye to grasp simultaneously the fundamental conception underlying the arrhythmias and thus simplify their study. In this manner, also, once the fundamentals of graphic curves is understood, and the corresponding abnormal activity of the heart is *visualized*, the student will the more easily apply these lessons at the bedside; for as will be shown (Chapter XI) the great majority of arrhythmias can be readily diagnosed without the use of either the polygraph or electrocardiograph. This can scarcely be done, however, unless the graphic basis of the arrhythmias is first thoroughly grasped.

The passage of the normal impulse, as exemplified in polygraphic and electrocardiographic tracings of the normal rhythmic cardiac mechanism, has already been described. We shall now discuss those abnormal mechanisms known as cardiac arrhythmias and irregularities. A. E. Cohn has succinctly grouped the arrhythmias as coming under variations of a few fundamental normal functions. He states that cardiac irregularities arise from the abnormal passage of the impulse, from abnormal sequence of contraction of the pairs of chambers, and from abnormal coordination of the muscle mass. So far as possible, these basic considerations have been incorporated into my Tabulation of the Arrhythmias. As will be seen, I have grouped all the arrhythmias either as arising in the auricle, in the ventricle, or in the specialized tissues, namely, the sino-auricular and the auriculo-ventricular nodes.

TABULATION OF TYPES OF ARRHYTHMIAS AND CARDIAC IRREGULARITIES

| | | |
|---|---|---|
| A. Auricular arrhythmias | I. Auricular extrasystoles.* | from the normal site. |
| | | from an abnormal site (ectopic). |
| | II. Paroxysmal tachycardia of auricular origin. | |
| | III. Auricular incoordination. | (1) Auricular fibrillation. |
| | | (2) Auricular flutter. |
| | | (3) Incoordination intermediate between flutter and fibrillation. |

* Because of its more common usage the term extrasystoles is here employed instead of "premature contractions." Both terms, however, may be used interchangeably.

- A. Nodal extrasystoles.
- B. Ventricular arrhythmias.
  - I. Ventricular extrasystoles (from right or left ventricle).
  - II. Interpolated extrasystoles.
  - III. Automatic ventricular activity (ventricular escape).
  - IV. Paroxysmal tachycardia of ventricular origin.
  - V. Ventricular incoordination.
    - (1) Ventricular fibrillation.
    - (2) Branch-bundle lesions.
- C. True bradycardia.
  - I. Disturbance in the sino-auricular node.
    - (1) Sinus arrhythmia or irregularity.
    - (2) Sino-auricular block (sinus block).
    - (3) Blocked auricular beat.
- D. Arrhythmias produced by abnormal sequence of contraction of auricles and ventricles.
  - II Disturbance in atrio-ventricular node.
    - (1) Prolonged conduction time.
    - (2) Shortened conduction time.
    - (3) Backward conduction.
    - (4) Auriculo-ventricular heart block.
      - (*a*) Incomplete heart block.
      - (*b*) Complete heart block (dissociation).

It is the aim of graphic methods to exactly transcribe the cardiac mechanisms, normal and abnormal. It has always been the aim of progressive clinical medicine to accept and adapt the knowledge gained by the use of exact instrumental methods for immediate use at the bedside, where instruments may not be available. It is thus that, in the light of knowledge gained by a careful study of the graphic methods, and with a full appreciation of the physiological pathology involved, it is possible to diagnose most types of cardiac and pulse irregularities by ordinary methods of examination. For this purpose, the stethoscope should be placed over the cardiac apex and the fingers kept on the pulse so that cardiac beats that are not propagated as pulse waves (so-called missed beats or pulse deficit) may be recognized. The neck should at the same time be carefully scrutinized for jugular and carotid pulsations (*a* and *c* waves). The value of keen observation will be discussed in connection with the clinical recognition of the arrhythmias.

## A. I. AURICULAR EXTRASYSTOLES

Regarding the general nature of extrasystoles—auricular, ventricular and nodal—some preliminary observations are required before their various types, and instrumental and clinical recognition are discussed.

Contractions of auricle or ventricle which anticipate the normal rhythmic time of their occurrence and disturb the normal rhythm are known as extrasystoles or premature contractions. Fundamentally, the normal passage of the impulse is disturbed. Extrasystoles were formerly termed "pulsus bigeminus." They are now sometimes popularly termed "dropped beats." As will be later pointed out, this is frequently a misnomer, for the beats are not actually "dropped;" they simply are at times not sufficiently powerful to lift the aortic cusps and produce a propagated pulse wave to the wrist. The term "premature contraction" is preferable because it so aptly describes the phenomenon. Extrasystole is also in a sense a misnomer, for the contrac-

tion is not "extra" or additional; it is simply anticipatory. However, since "extrasystole" is commonly used, it will be here employed. If extrasystoles recur at regular intervals after each normal beat, the resultant rhythm is known as coupling, coupled rhythm, or coupled beats. There are certain characteristics, as Lewis has pointed out, which physiologically differentiate the premature from the normal contraction. The normal physiological (or homogenetic) beat is one of a series of similar normal rhythmic contractions; the contractions are equally spaced; there is an orderly building up of impulse formation which requires at least one half second for each beat.

FIG. 106.

FIG. 107.

FIG. 108.

FIG. 109.

FIG. 110.

FIG. 111.

FIGS. 106–111.—Diagrams illustrating the origin of physiological and pathological contractions.

FIGS. 106–107.—S.A., Sino-auricular node; A.V., part of the atrioventricular conduction system.

FIG. 106.—X.X., Origin of homogenetic beat in the sinus region with normal propagation in the junctional tissue.

FIG. 107.—X, Ventricular extrasystole.

FIG. 108.—X, Auricular extrasystole.

FIG. 109.—X, Nodal extrasystole.

FIG. 110.—Disturbance in rhythm from a premature auricular contraction.

FIG. 111.—Disturbance in rhythm from a premature auricular contraction.

This power of rhythmic impulse formation resides chiefly, if not entirely, in the specialized tissue of the sino-auricular node, of the atrio-ventricular node, or in the bundle of His (Fig. 106). Regarding the premature, extrasystolic, heterogenetic, or pathological contraction, (terms used here synonymously),

whether single or multiple, they are exceedingly rapid and abrupt; indeed there may be no time for impulse formation. Ventricular extraystoles, for example, may follow each other at intervals as short as 0.25 second or even less. Extrasystoles are further differentiated from normal beats by their prematurity and by their lack of rhythmic tendency; even when multiple or when occurring in showers, the rate of production is usually maximal. Further, there is no relationship between the heightened activity of the physiological heart rhythm and the prevalence of extrasystoles. Often, influences which depress the one favor the occurrence of the other; such an example is

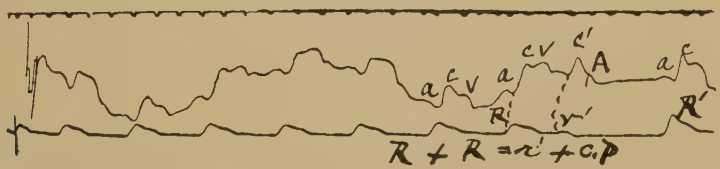



FIG. 112.—R,[1] Rhythmic beat; C.P., compensatory pause; $r'$, extrasystole; R', larger post extrasystolic beat. In the jugular tracing, the premature wave $c'$ is synchronous with $r'$. Its foot-point is determined by measuring off with dividers the distance $Rr'$ from the preceding rhythmic $c$ ($Rr' - Cc'$). The auricular beat $A$ is not premature. It falls at its rhythmic time; hence, the inter-auricular distance $a - A'$ is equal to the normal beat ($R$). A occurs before the $c'$ wave has completed its fall, hence the abnormal width of the combined $c'A$. The A wave is often not indicated as a distinct part of the $c'A$ because it may be lost in the more prominent $c'$ wave.

[1] In all the polygraphic tracings the time-marker measures 1/5 second.

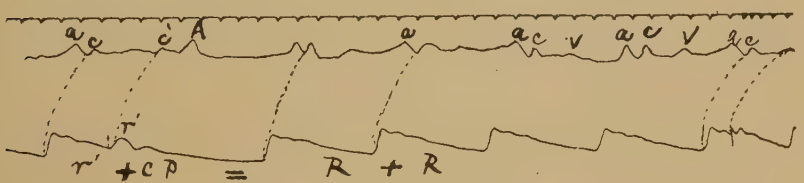



FIG. 113.

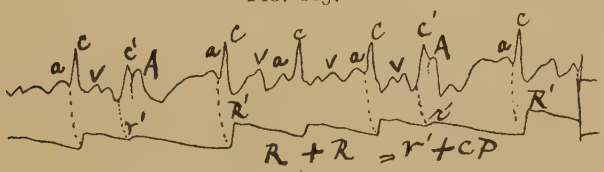



FIG. 114.

FIGS. 113, 114.—Ventricular extrasystoles with typical compensatory pauses.

chloride of potassium in the experimental animal. Premature contractions bear a different relationship to the cardiac nerves than does the normal beat; for instance, gradually increased right vagus stimulation retards the normal rhythm; it has no effect on, or produces abrupt cessation in, a series of premature contractions. Extrasystoles rarely originate from the normal rhythmic center, a fact electrocardiographically shown by differences in their form. Finally, the nearer the origin of the normal physiological beat to the vicinity of the superior vena cava, the faster the rhythm. This law does not

apply to extrasystoles; their rate does not depend upon their origin in auricle or ventricle.

Premature beats (Figs. 107, 111, 112), because of their inherent weakness and because the blood may not be properly directed against the aortic cusps,

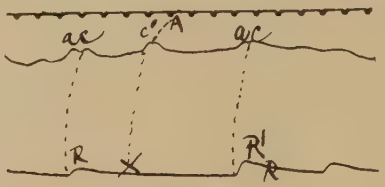



FIG. 115.

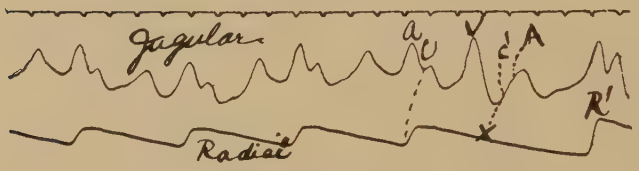



FIG. 116.

FIGS. 115, 116.—Ventricular extrasystoles with *c'* in the jugular but no representation in the radial tracing. X marks the point where the extrasystole should have produced a pulse wave.

are occasionally "frustrane" or "abortive;" that is, they either do not open the aortic cusps at all or do so insufficiently; hence in either instance, they fail to produce a propagated radial wave. These frustrane contractions are

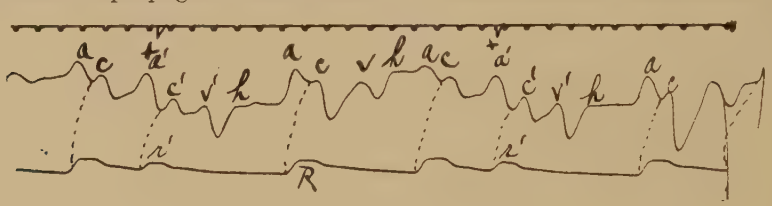



FIG. 117.

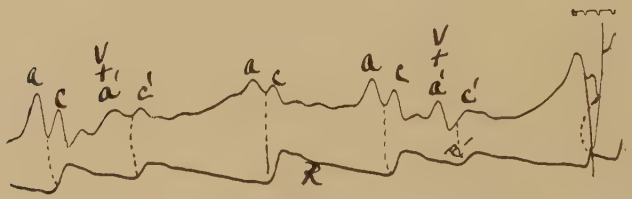



FIG. 118.

FIGS. 117, 118.—Auricular extrasystoles. The auricular beat *a'* is premature and is followed by the *c'* wave. The *v* wave of the rhythmic beat has fallen with *a'*.

sometimes popularly termed "missed" or "dropped" beats, an evident misnomer, because ventricular contractions do actually occur, but the blood is not propagated as a pulse wave for the simple mechanical reasons above described. Yet I have found much confusion among students and practitioners regarding this simple fact in physics. It is therefore worth repeating that the only

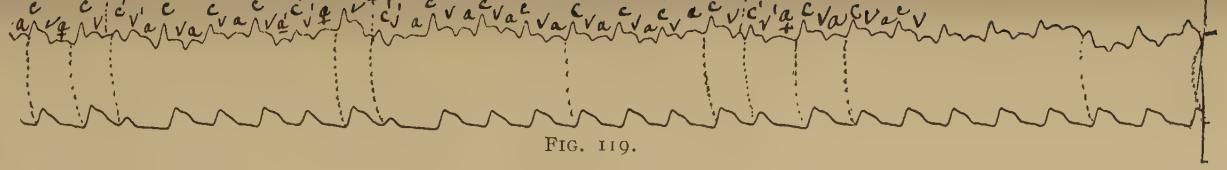


FIG. 119.

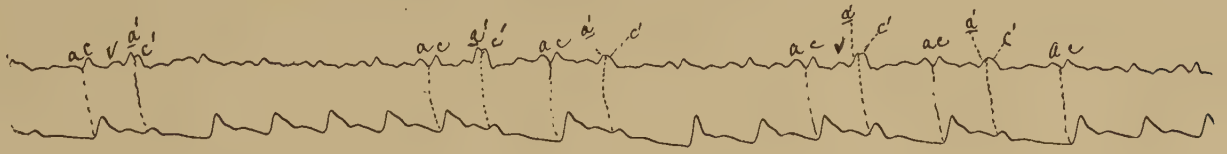


FIG. 120.

FIGS. 119–120.—Auricular extrasystoles with shortened conduction time ($a'c'$). There is lengthened conduction time ($a$–$c$ interval) in some of the rhythmic beats.

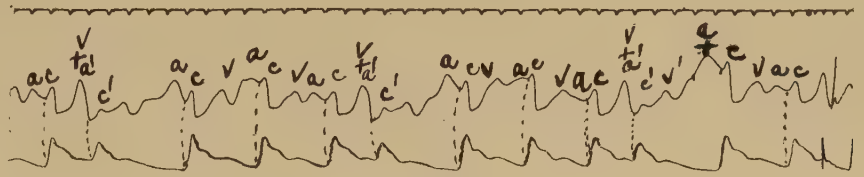


FIG. 121.

FIG. 121.—Auricular extrasystole ($a'c'$). Lengthened conduction ($a$) of the rhythmic beat.

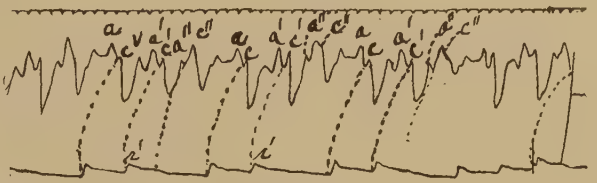


FIG. 122.

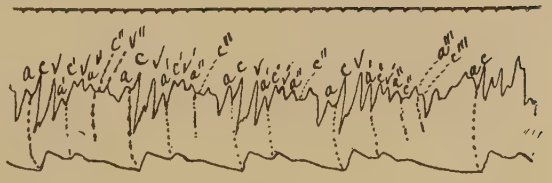


FIG. 123.

FIGS. 122 123.—Multiple auricular extrasystoles. The radical tracing resembles coupled rhythm, but the frustrane contractions are well seen in the jugular tracing ($a''c''$, $a'''c'''$).

Fig. 124.—Auricular extrasystoles.

reasons an extrasystole may be heard or felt at the heart and not always felt at the wrist are two—The premature contraction is too weak to open the aortic cusps sufficiently to push the blood along the systemic circulation; or the blood is not directed towards the aortic cusps and hence blood is not ejected into the systemic circulation. Not only may abortive contractions escape palpation, they may be even too minute to be seen in radial tracings. In such instances the phlebogram shows the usual evidence of premature contractions (Figs. 115 c, 116 c). The method of seeking the point of incidence of the rhythmic auricular beat has already been shown (*a* wave, Figs. 112, 113, 114). Abnormal contractions, single or multiple, that originate outside of the sinus region are sometimes technically called "ectopic" contractions (Lewis). Auricular extrasystoles are usually "ectopic" in this sense, but this fact can only be demonstrated electrocardiographically by the difference in the form of the abnormal auricular complex (P waves, Fig. 128) from the normal; that is to say, in any tracing the normal electrocardiographic P wave derived from the normal pace-maker site (the sino-auricular node) has a different form from that coming from any abnormal auricular point of origin. Auricular extrasystoles are not necessarily premature in their occurrence and hence may not disturb the normal rhythm. This statement also applies to any type of extrasystoles, for, while usually premature, they occasionally occur at the time of, or even shortly after the time the normal beat should occur.

When auricular extrasystoles are present, the ventricle usually responds to the premature auricular contraction after normal conduction time; this means that there is no delay or shortening of the interval required for the impulse to travel from the auricular extrasystolic contraction to the ventricle.

As may be seen in the polygram, auricular extrasystoles produce radial waves of varied

size and strength (r, Figs. 117, 118); frustrane contractions (that is, those that produce no pulse waves) are comparatively rare. The auricular

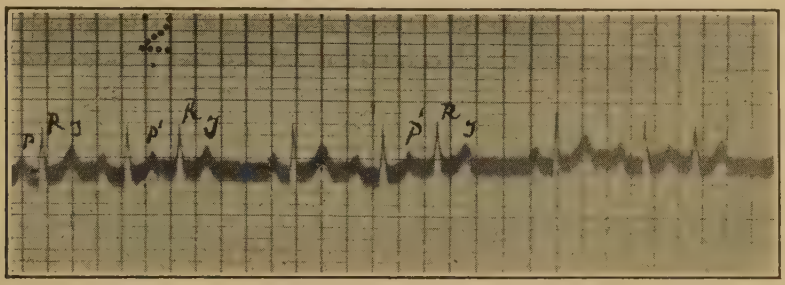


FIG. 125.—Auricular extrasystoles. The conduction time (P'R') is slightly prolonged.

extrasystole in the jugular tracing is usually marked by normal conduction time between it and the answering ventricular beat (a, c, Figs.

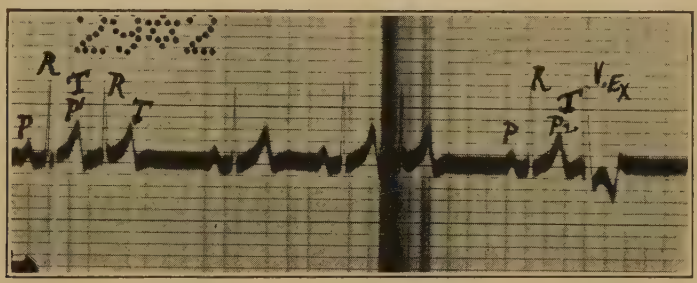


FIG. 126.—Auricular extrasystoles superimposed upon the T wave (TR'). The premature auricular contraction P2 is followed by an ectopic ventricular beat (V.Ex); *i.e.*, the latter has followed a course in the ventricle different from the rhythmic beats (ventricular extrasystole).

117, 118); the conduction time however, is occasionally diminished (a, Figs. 119, 120) or increased (Fig. 121). Extrasystoles are sometimes multiple (Figs. 122, 123). If they are registered in the radial curve, the latter resembes coupled rhythm; the jugular tracing will then reveal the frustrane extrasystoles (Figs. 122, *a*, *c*, 123, *a'*, *c'*, *a''*, *c''*,) which have produced jugular but no radial waves.

When auricular extrasystoles originate in the pacemaking area, *i.e.*, at the normal site, the electrocardiograms are identical in shape with those of the normal; the only distinguishing feature is their prematurity. The ventricle usually responds after a normal P-R interval, (the measure of the conduction time in electrocardiograms), and its complex is the same as the other rhythmic beats because it follows the normal path in the conduction system (Figs. 124,

FIG. 127.—Auricular extrasystole showing the superposition of P and T waves (P'T').

125, 126). The premature auricular contraction often falls at such time that its complex is superimposed upon the T wave (Fig. 127), so that upon superficial examination the P wave (auricular beat) appears absent. Careful scrutiny, however, indicates that the composite P-T wave is somewhat taller and thicker than the P wave alone (Fig. 126). As has been stated, "ectopic" auricular contractions which originate in an abnormal site outside the pacemaker show their origin by change of complex. This is exemplified in Fig. 128, which demonstrates beats starting from different ectopic foci (A, Ex. 1; A, Ex. 2); these abnormal complexes are deviated upwards and resemble the normal. This probably indicates an origin close to the pacemaker. It is to be noted that in this instance the ectopic beats are neither premature nor are they followed by compensatory pauses; the only feature differentiating them from the normal is their slight difference in form from that of the rhythmic P wave.

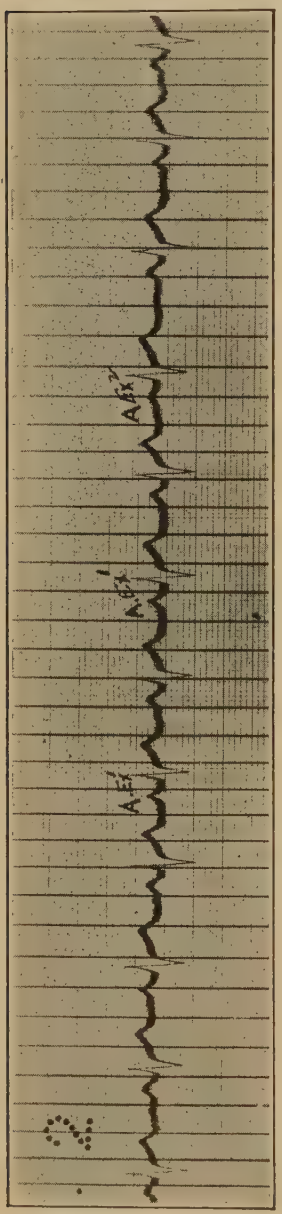



FIG. 128.—Auricular extrasystole coming from two ectopic foci, A.E X. 1, A.E X. 2. These beats are neither premature nor followed by compensatory pauses. Their ectopic origin is denoted by the difference in form from the normal rhythmic complex (P wave).

**Clinical Recognition of Extrasystoles (Premature Contractions).**—It should be emphasized that arrhythmias, especially extrasystoles and paroxysmal tachycardia, are often transient; hence they may not be present at the time patients present themselves for examination. As will be pointed out, however, the patient's description of his sensations is usually so clear and vivid, that the physician can often venture not only the diagnosis of a cardiac irregularity, but even its type. On the other hand it should be remembered that irregularities do not always cause subjective sensations. Here naturally the diagnosis must be made by the physician's objective examination.

Only occasionally can a clinical differentiation between auricular and ventricular extrasystoles be made without graphic tracings. Hence, in what follows, the recognition of either type is meant except when otherwise stated.

Isolated extrasystoles, occurring once in several normal beats, and followed by long (compensatory) pauses are readily diagnosed. When interpolated (q. v.) or when occurring in showers (multiple extrasystoles), their recognition is more difficult. If the premature contraction is frustrane, that is, if its course in the ventricle is improperly directed or the beat itself is not sufficiently powerful to open the aortic valves, there is no corresponding pulse beat at the wrist; otherwise, the extrasystole causes a stronger or weaker pulse beat. Depending entirely upon the mechanics of intraventricular pressure affecting the aortic cusps, one or two cardiac sounds of varying intensity accompany the premature contraction. If the extrasystoles open the aortic valves, and the latter subsequently close, a premature first and a premature second sound accompany the extrasystole, just as with a normal beat. These sounds are however fainter than those of the normal beat. If the ventricular pressure remains lower than the aortic, a premature first sound alone is heard. Only rarely is this sound too faint to be audible. While graphic methods of registration are required to determine the exact length of the compensatory pause (Fig. 112), distinct shortening of the latter is readily determined by palpation at the wrist. Marked differences in the duration of the compensatory pauses characterize auricular, rather than ventricular, extrasystoles. The likelihood of a premature contraction being auricular is increased if it produces a fairly strong pulse beat; the weaker or absent pulse beat is more characteristic of a ventricular premature contraction. It is occasionally possible to distinguish auricular from ventricular extrasystoles by observation of the jugular pulsations, for in the ventricular type a large summation wave (corresponding to the polygraphic *a* and *c* waves, Fig. 112) may occasionally be seen.

**Coupled Rhythm.**—Its diagnosis rests upon the clinical recognition of regularly recurring extrasystoles, that is, an extrasystole occurs after every rhythmic beat. Sometimes a type of coupling occurs in auricular fibrillation, especially as the result of digitalis medication. The distinction from the coupling just described can only be made from observation of the clinical characteristics of auricular fibrillation (q. v.).

Extrasystoles occurring at irregular times and with varying force in a rapidly beating heart are most apt to be confused with auricular fibrillation, for, like the latter the heart and pulse are apt to be confusedly irregular. The clinical differential guide is observation of the jugular and of carotid beats to find out whether one can distinguish normal auricular and carotid waves in the neck; even then, differentiation is often impossible because of the rapidity of the pulsatile waves which makes it extremely difficult for the eye to decipher them.

### A. II. PAROXYSMAL TACHYCARDIA OF AURICULAR ORIGIN

The term simple tachycardia should be applied to the common acceleration of the normal sinus rhythm, that is, to the usual types of rapid heart

action. The usual ventricular rate is then between 110 and 130 per minute. Paroxysmal tachycardia of auricular origin (paroxysmal auricular tachycardia) as shown by the electrocardiogram usually consists of auricular extrasystoles coming in attacks. In typical instances, there is extreme, abrupt acceleration of the normal rhythm, with an abrupt termination and return to the normal. The attack may last minutes, hours, or, more rarely,

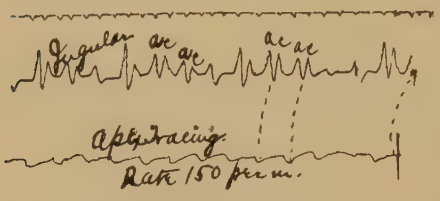



FIG. 129.

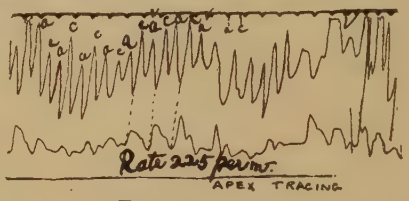



FIG. 130.

FIGS. 129, 130.—Varying ventricular rates from a case of exophthalmic goiter recorded on different days. When the rate is 150 per minute (Fig. 129) the *a-c* interval is normal. When the rate is 225 (Fig. 130), the *a-c* interval is slightly diminished; the diastole is entirely abolished, the *a* wave falls with the *v* of the preceding beat.

days. Like the single auricular extrasystole, it is basically due to the fact that the excitation wave and impulse follow an abnormal passage through the heart. Graphically studied, the attack is seen to begin with a definite "onset" and to end with a definite "offset." Typical attacks are initiated

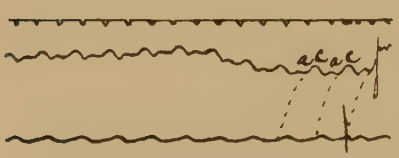


FIG. 131.—Paroxysmal tachycardia. rate, 130 per minute. Onset and offset not recorded. The *a* wave falls with the preceding *v*.

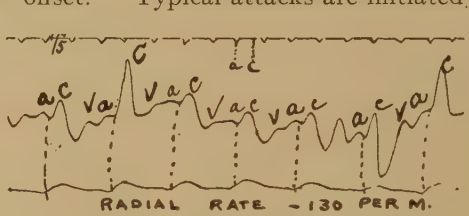



FIG. 132.—Exophthalmic goiter. Ventricular very much shortened conduction time (*a-c* interval) despite only moderate acceleration (simple tachycardia).

and terminated by extrasystoles. The ventricular rate usually varies from 170 to 200 per minute. The rapid heart action of simple and paroxysmal tachycardia is usually at the expense of diastole (Figs. 129–131). The conduction time as well is occasionally diminished (Figs. 132–135). In some cases of exophthalmic goiter, I found diminished auriculo-ventricular conduction time despite the fact that ventricular acceleration was not extreme (Figs. 132–135).

Studied electrocardiographically, it is found that the paroxysms commonly originate from an abnormal ("ectopic") auricular focus. When the change in the auricular complex is not marked, an origin close to the normal pacemaker is assumed. When the auricular complex (the P wave) is deviated negatively, it indicates an origin in the lower part of the auricular musculature.

**Clinical Recognition of Simple Tachycardia and of Paroxysmal Tachycardia of Auricular Origin.**—The former refers to rhythmic pulse and ventricular action at the rate of about 120 or more per minute. Its recognition is simple in the vast majority of cases. It may be confused with the rapid heart action of auricular fibrillation, but the pulse of the latter will be found arrhythmic upon longer or shorter observation. Paroxysmal (auricular) tachycardia is also readily recognized by the rapid, regular heart action vary-

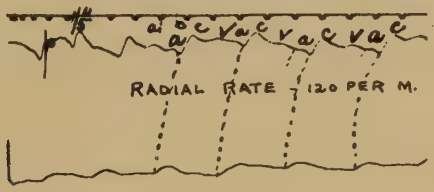



FIG. 133.

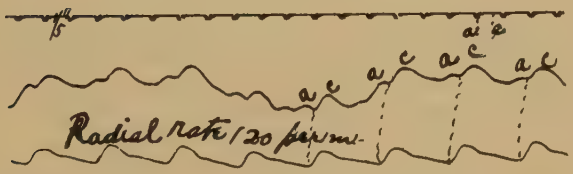



FIG. 134.

FIG. 133, 134.—Exophthalmic goiter. Ventricular rate 120 per minute; simple tachycardia; shortened *a-c* interval.

ing from 170 to 200 per minute, accompanied by a feeble and rapid pulse; by the regular rapid jugular pulsations in the neck, and by the characteristic onset and offset of the paroxysm, for an extrasystole with its compensatory pulse often initiates and completes the typical attack. During the attack rapid, regular heart action is so constant that the rate rarely varies more than two or three beats from minute to minute. Hence if the heart rate is counted carefully for an entire minute and then again for an entire minute a few minutes thereafter, the cardiac rate for both observations will be found to be almost the same. This is another simple method of diagnosing paroxysmal tachycardia of auricular origin when the patient can be observed during the attack. Data or observation, however, often can not be obtained by direct clinical observation because the attacks are apt to be transient, and one may entirely miss the attack, or miss its beginning or termination. In most instances, therefore, we shall have to depend upon the history of sudden beginning and termination of the attack or upon the description of a sudden fluttering in the chest that begins or ends suddenly. In addition, we may obtain a fairly

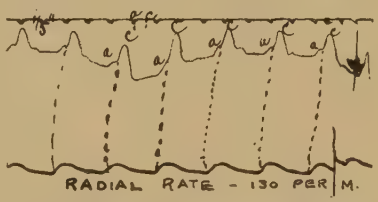



FIG. 135.—Exopthalmic goiter. Ventricular rate 130 per minute; simple tachycardia; *a-c* considerably decreased.

accurate description of the occurrence of an extrasystole by such accounts as: "The heart stops for a moment," or: "There is a momentary faint feeling in the chest before or after the palpitation," or: "My heart knocks or kicks before it beats fast." Intelligent and observing patients may give a sufficiently accurate account of the cardiac rate during the attack by asking them to tap with their fingers approximately as rapidly as they thought their heart was "palpitating."

The differentiation of paroxysmal tachycardia from auricular flutter (q. v.) and from the tachycardial attacks accompanying auricular fibrillation (q. v.) will be discussed under those arrhythmias.

### A. III. AURICULAR INCOORDINATION

It will be noted that the three types of arrhythmia to be described—fibrillation, flutter and the intermediate form—result primarily from auricular incoordination, hence this caption. An extreme degree of inchoate auricular activity results in auricular fibrillation. When incoordinate activity is less marked, it results in auricular flutter (also called auricular tachysystole).

### A. III. (1) AURICULAR FIBRILLATION

A correlation of various statistics shows that auricular fibrillation comprises approximately 40 per cent. of arrythmias from all causes. We know now that the great majority of cases which Mackenzie called nodal rhythm belong to this category. When auricular fibrillation is typical, ventricular activity is completely irregular in rhythm and force so that no two successive beats are alike. When induced experimentally in animals, the auricles show irregular tremulous fibrillating activity; sometimes and in some parts of the auricles the fibrillation is fine, and at other times and other places it is coarse. There is no uniformity in auricular contractility, although occasionally waves of fibrillation proceed with a fair degree of regularity over the entire musculature. The auriculo-ventricular conduction system is bombarded, as it were, by numerous irregular impulses, only some of which can pass through the junctional tissue and excite the ventricle to irregular and discordant response.

The polygram corresponds to the pathological physiology just described. In typical instances the radial tracing shows gross and complete irregularity in the force and rhythm of the radial beats; in other words, a grossly irregular pulse. In the jugular, the representative of orderly rhythmic auricular contraction, the *a* wave, is absent (Figs. 136–150). It is said that fibrillation is sometimes sufficiently coarse to produce small fibrillary reflux waves in the superior vena cava and jugular bulb; these are then indicated in the tracing as irregular wave-like lines. When found in mitral stenosis with a marked diastolic thrill, it seems to me that such fibrillary waves may be due,

FIGS. 136–149.—Different types of polygrams of auricular fibrillation. The radial tracings show varying degrees of irregularity of rhythm and force of the pulse beats; some are grossly irregular (for example Figs. 137, 143, and 146); others more nearly approach the normal pulse rhythm (Figs. 142, 147, 149). The jugular tracings are of the most varied types. Their pathognomonic characteristic is the absence of the rhythmic *a* wave regularly preceding the *c*. Small waves preceding the *c* are sometimes observed (Figs. 136, 137 and 139), but their incidence and size are irregular. The *c* wave may be split (Figs. 144, 148) or may form a combined wave with the *v* (Figs. 147 and 149), especially when ventricular action is slow.

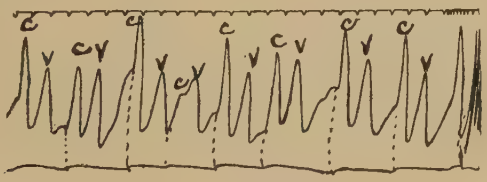


FIG. 136.

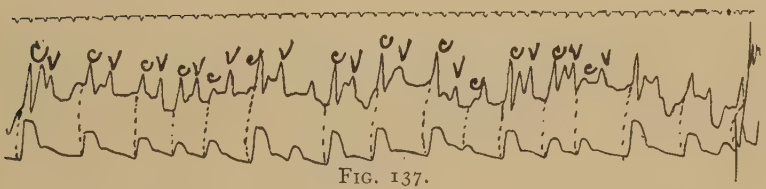


FIG. 137.

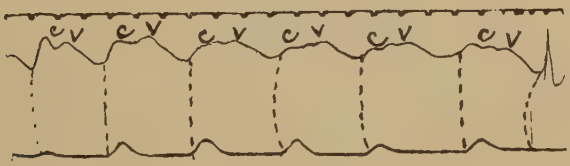


FIG. 138.

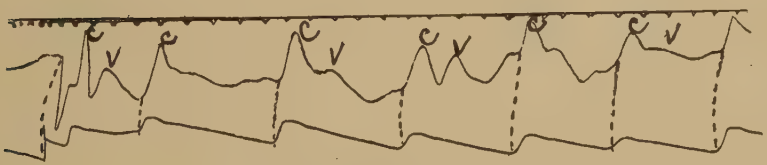


FIG. 139.

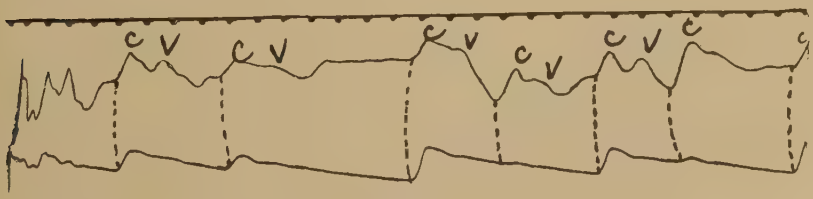


FIG. 140.

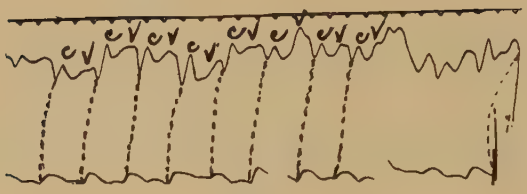


FIG. 141.

FIG. 142.

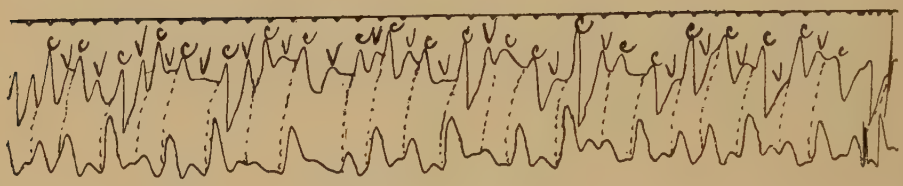


FIG. 143.

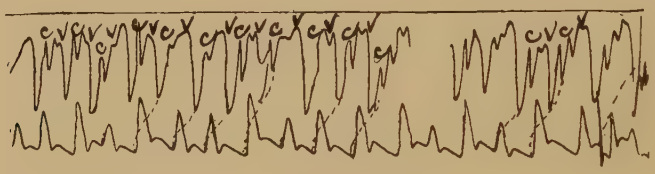


FIG. 144.

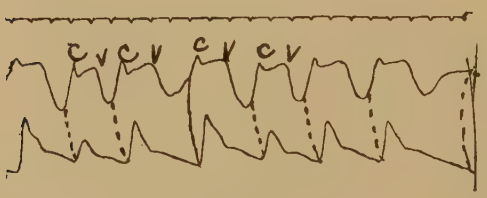


FIG. 145.

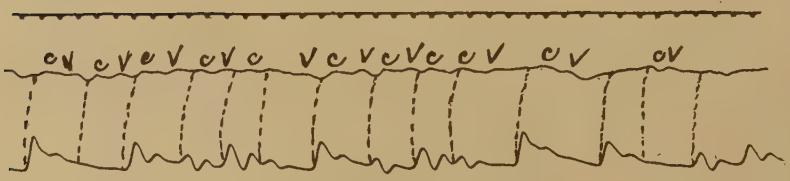


FIG. 146.

FIG. 147.

FIG. 148.

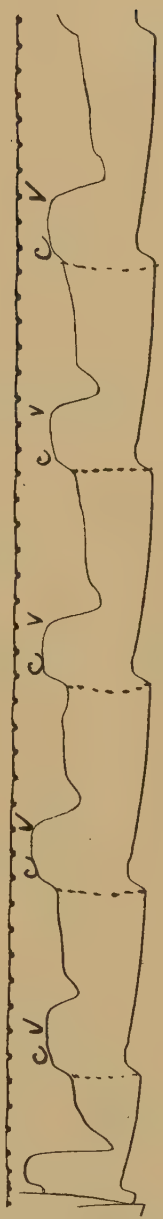


FIG. 149.

not to auricular fibrillation *per se*, but to turbulent diastolic ventricular eddies propagated as small waves from vibrations of the stiffened mitral valves, and transmitted directly through the heart muscle to the superior vena cava and jugular veins. Figure 151 shows fibrillary waves from a case

FIG. 150.—Auricular fibrillation, anacrotic radial pulse.

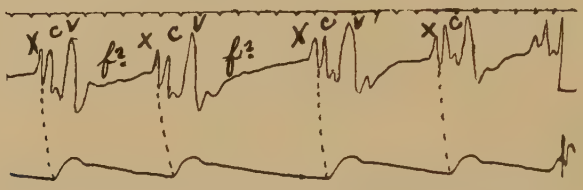


FIG. 151.—Auricular fibrillation with mitral stenosis showing presystolic (*x*) and fibrillary (*f*) waves.

of mitral stenosis with a loud, exceedingly rough, diastolic murmur and thrill. There was in addition, a short, sharp, regularly recurring presystolic wave (Fig. 151, X) whose etiology is not clear. Electrocardiograms taken at that time preclude the possibility of its being an auricular wave.

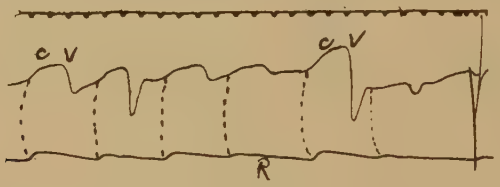


FIG. 152.

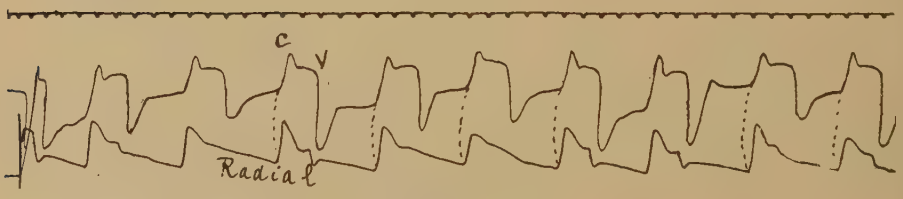


FIG. 153.

FIG. 152 and 153.—Auricular fibrillation; sharp fall of pressure following the ventricular filling wave. In Fig. 153, the radial pulse is quite rhythmical. Digitalis had been given.

In some jugular tracings of auricular fibrillation, a sharp rise and subsequent fall of pressure succeeding the *v* wave is found (Figs. 152, 153). As already indicated, a completely irregular pulse accompanies auricular fibrilla-

FIG. 154.

FIG. 155.

FIG. 156.

FIG. 157.

FIG. 158.

FIGS. 154–158.—Auricular fibrillation illustrating the usual types of accompanying pulse irregularities.

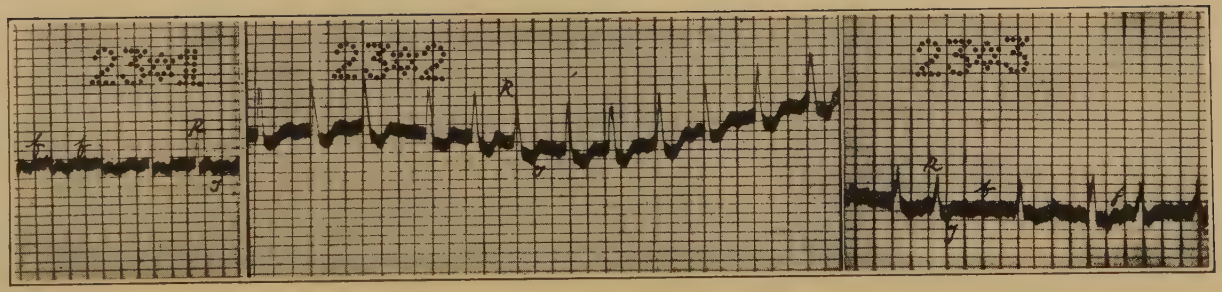


FIG. 159.—Small, fairly regular fibrillary (*f*) waves in parts of leads I and III.

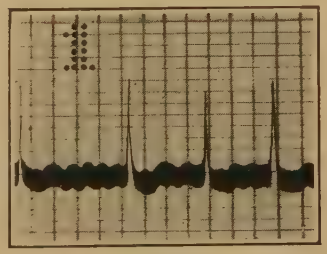


FIG. 160.—Fibrillation waves scarcely visible.

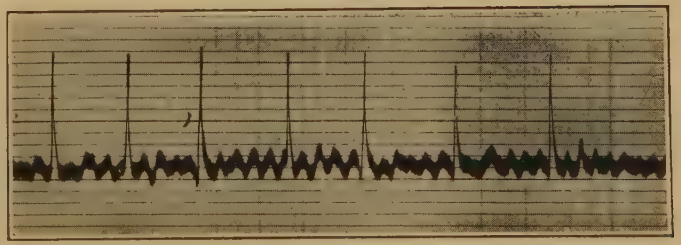


FIG. 161.—Fairly regular fibrillation waves which at times resemble auricular flutter (q. v.). (Impure flutter).

tion in a great majority of cases (Figs. 154–158). Very rarely the pulse becomes absolutely regular as the result of digitalis medication, although fibrillation still continues (Fig. 153).

Corresponding to the state of incoordinate hap-hazard auricular activity characteristic of auricular fibrillation, there is in the electrocardiogram an absence of regularly recurring auricular deviations; that is, P waves are

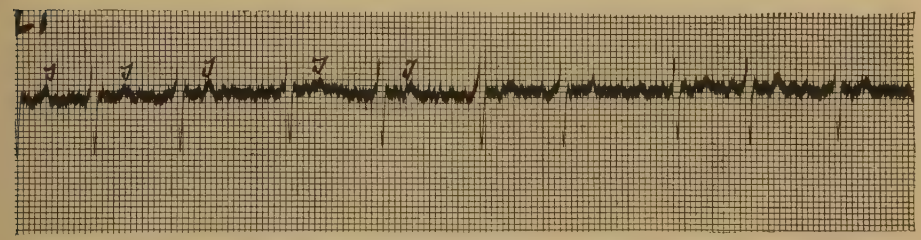


FIG. 162.—Fine fibrillation waves. Note the absence of P waves.

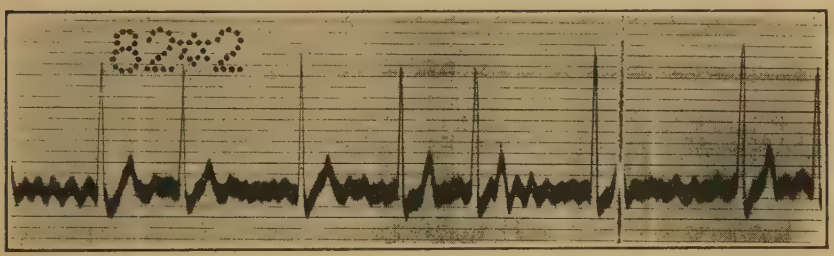


FIG. 163.—Various types of fibrillation waves, coarse and fine, in the same lead.

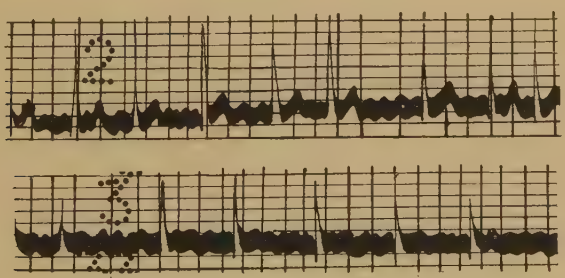


FIG. 164.—Fairly regular fibrillation waves in parts of L II and L III.

missing. In their place, there are undulations representing fibrillation, at rates between 350 and 900 per minute. These fibrillatory waves vary considerably in type. In short runs they may resemble the regular and rapid auricular activity of flutter (impure flutter); at other times the waves are coarse and arrhythmic, or are so small as to be scarcely distinguishable as separate deviations. Examples of these various types are shown in Figs.

159–167. At present there is no clinical distinction based upon these differences, except possibly in their reaction to quinidin (Chapter XX).

In older people with cardio-sclerosis, or as a result of digitalis medication, the pulse and heart action may be fairly regular in force and rhythm,

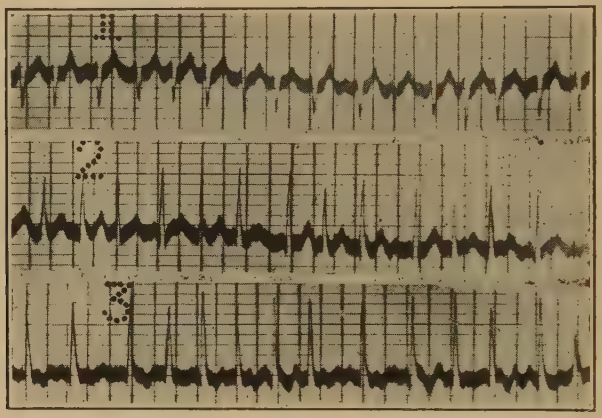


Fig. 165.—Auricular fibrillation showing tachycardia in L I. Note the varying rates of ventricular rapidity in leads II and III.

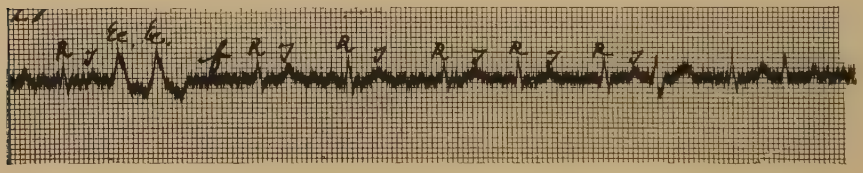


Fig. 166.—Auricular fibrillation with ectopic beats (extrasystoles, Ec.).

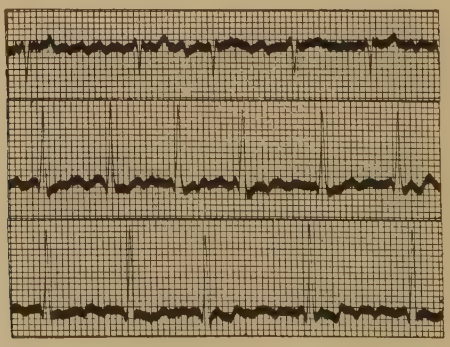


Fig. 167.—Auricular fibrillation with coarse fibrillation. (*Courtesy of A. E. Cohn.*)

so that graphic tracings are required in order to establish the diagnosis of fibrillation. In another clinical type of fibrillation, the pulse rate varies between 180 and 225 per minute, is small in volume and is easily compressible.

Upon palpation, the individual beats appear equal in force. Clinically, this resembles paroxysmal tachycardia. Electrocardiographically, it is distinguished from the latter by the absence of P waves and by the absence of an "onset" and "offset" (Fig. 165).

Abnormal "ectopic" beats are sometimes present in auricular fibrillation (Fig. 166, Ec.). They are erroneously called extrasystoles; the latter term is properly applicable only to premature beats which disturb an otherwise regular rhythm, and not to beats occurring in the complete irregularity of auricular fibrillation.

**Transient Auricular Fibrillation.**—Only exceptionally is it possible to note sudden transitions from normal rhythm to auricular fibrillation. Such attacks may be the result of digitalis therapy. They are also found in exophthalmic goiter or in the crises of severe infections (for example, in pneumonia). They are by no means infrequent in older patients with cardiosclerosis in whom hypertension is a marked clinical feature (q. v. Carcio-Vascular Clinics). Attacks of auricular fibrillation, in addition, may occur in the decompensatory stages of all types of valvular disease, especially mitral stenosis. Its occurrence with auricular flutter has already been pointed out. Its presence in a patient with no temperature and with no sign of organic cardiovascular disease is very rare. Figure 168 is an example. It was taken from a child of eight, not neurotic, who had had several attacks of tonsillitis. Tonsillectomy had been performed two years previously. Shortly before she came under my observation, she suddenly felt her heart "jump:" this "jumping" has since been occasionally repeated. There were no gastric symptoms. The child was somewhat anemic and undersized. There were no signs of decompensation. The urine contained no abnormal elements. There was a very soft faint systolic murmur of functional nature at the apex; the murmur was not transmitted. The electrocardiogram (Fig. 168, L III) shows the above-mentioned short run of auricular fibrillation (A.F. . . . A.F.) following the ventricular extrasystole (V.Ex.). In addition, there is fairly marked sinus arrhythmia. There are several ventricular extrasystoles, (V.Ex.) in some of which the auricular beat is seen as a separate deviation (P). There is also an auricular extrasystole of sinus origin with an abnormal ventricular complex (L I, A.Ex.); which is not followed by a compensatory pause.

**Clinical Recognition of Auricular Fibrillation.**—The pulse and ventricular irregularity of typical auricular fibrillation is sufficiently obvious to be readily manifest. At the apex there is marked *irregularity in force and rhythm* of the heart's action. Sometimes scarcely two beats are alike. Indeed, the old term of a generation ago—*delirium cordis*—aptly describes the clinical impression. Sometimes there is fairly regular heart action lasting several seconds or longer. At the wrist, if all the cardiac beats are propagated and come through as pulse waves, the pulse, completely irregular, takes on a helter-skelter characteristic. In the neck, the carotids are correspondingly

FIG. 168.— Various parts of a continuous electrocardiogram. The leads are indicated by the numbers. Several ventricular extrasystoles (Ex), some showing distinct auricular beats (P'), are seen. An auricular extrasystole of sinus origin and followed by an abnormal ventricular complex is shown in L I (A.Ex.). In L III a short run of auricular fibrillation (AF-AF) followed by a ventricular extrasystole (V.Ex.) is shown.

irregular; the jugular pulsations are usually too rapid to be individually identified. If there are many frustrane ventricular contractions (so-called pulse deficit), the radial seems fairly regular in force and rhythm, for it is mainly the small and weaker beats which produce the picture of complete irregularity: Ausculation at the cardiac apex will readily reveal these small, numerous, discordant beats. Indeed, in thin persons, mere inspection of the apical region shows the characteristic arrhythmic ventricular action of auricular fibrillation.

The "ectopic" beats (erroneously called "extrasystoles"), occasionally present in auricular fibrillation, can sometimes be recognized even when ventricular action is rapid and irregular, because they are commonly followed by momentary (not compensatory) pauses, and by beats much louder and stronger than themselves. In the tachycardial attacks occurring with auricular fibrillation, there is usually no sudden beginning or termination; there is no onset or offset, the beats are sufficiently irregular in force and rhythm to be palpable and are apt to be interrupted with the typical gross irregularity of auricular fibrillation; after the attack, a completely irregular pulse is again present. These are data which serve in the differentiation between this type and the paroxysmal (auricular) tachycardia already discussed.

In auricular fibrillation with coupled rhythm, and fairly slow and regular ventricular activity and pulse, or in those rare instances of a perfectly regular pulse following digitalis medication, differentiation from normal rhythm or from extrasystolic arrhythmia can only be made by direct observation of the jugular pulsations. If distinct auricular waves can be recognized, their presence serves to exclude fibrillation.

### A. III. (2) AURICULAR FLUTTER—AURICULAR TACHYSYSTOLE

In this type the auricles beat regularly and rapidly from 225 to 350 times per minute. Since the ventricle cannot respond at a like rate, heart block (q.v.), incomplete or complete, results. If the block be incomplete at a 2:1, 3:1, or 4:1 ratio, it is evident that the pulse remains regular, the rate depending upon the ratio. If incomplete heart block be present with constantly varying auriculo-ventricular ratios, the pulse becomes irregular. The polygraphic recognition of auricular flutter may be difficult, for the auricular waves in the polygram are often small and distorted by respiration. Lewis contends that when the pulse is irregular in auricular flutter, the arrhythmic groups of radial beats form exact multiples, so that auricular flutter may be diagnosed from the radial curve alone. I have not found this to apply to some cases of flutter that I measured and attempted to diagnose in this manner. When auricular waves are well marked (Figs. 169, A and B, Fig. 170), flutter is readily recognized. In the interpretation of the degree of block, it must be remembered that, when incomplete, the *a-c* interval may be abnormally prolonged; this may then give the appearance of shortened

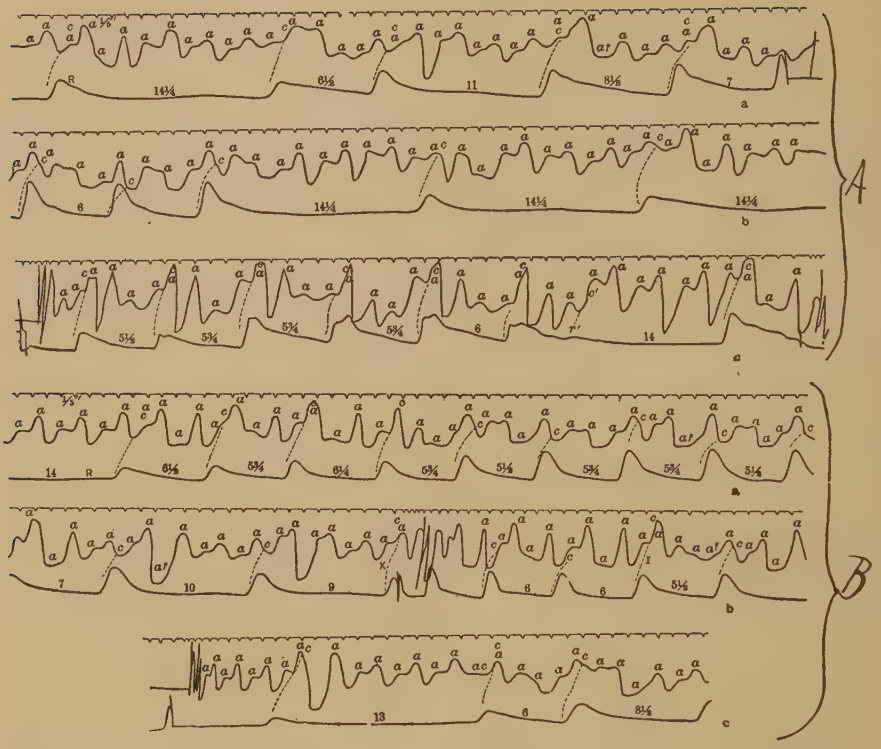



FIG. 169.—Tracing of auricular flutter. A and B were obtained on different days. *a*, *b*, *c* are continuous. In A, the auricular rate is 210; in B, 245 per minute. The numbers on the radial beats denote their respective lengths in multiples of $\frac{1}{5}$ seconds. The block is complete in A (*a* and *b*) and changes from incomplete to complete in *c*. The block is incomplete in B (*a* and part of *b*) and then later becomes complete.

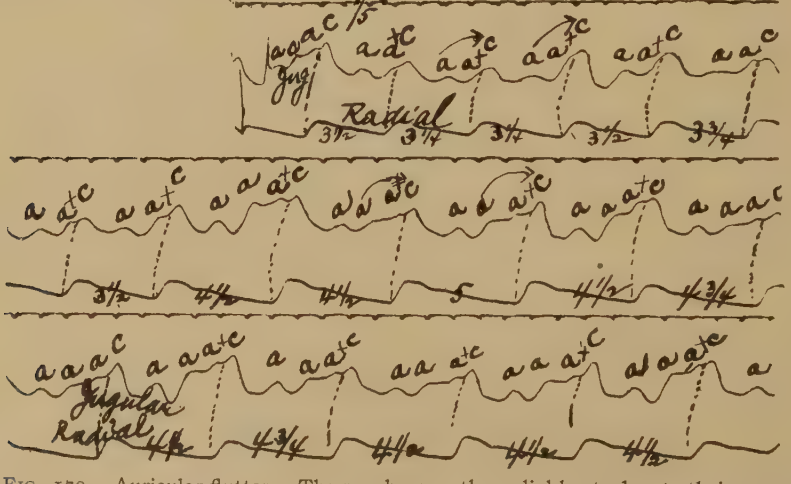



FIG. 170.—Auricular flutter. The numbers on the radial beats denote their respective lengths in multiples of $\frac{1}{5}$ seconds. The auricular impulse and its corresponding ventricular response are shown by arrows.

conduction time, for the ventricle has probably responded, not to the immediately preceding auricular beat, but to the one previous (indicated by arrows, Figs. 170, 171; the conduction time is approximately 0.4 second). Partial heart block with a 3 : 1 rhythm is shown in Fig. 173. The pulse is almost regular throughout, although there are shorter and longer beats indicative of change in auriculo-ventricular ratio. In other instances of flutter there may be found a change from partial to complete block, or the reverse (Fig. 169).

FIG. 171.—Auricular flutter—partial heart block. The occasional difference in the lengths of the radial are due to changes in the ratio of auriculo-ventricular rhythm or possibly to differences in conduction time.

An electrocardiographic example of auricular flutter with incomplete and complete block in the same electrocardiogram is shown in Fig. 172. Rapid, regular auricular activity is best seen in L II and III. The ventricular rate is 75; the auricular, 300 per minute; that is, incomplete block at a 4: 1 ratio was present. The P and T waves are occasionally superimposed. The P waves are contiguous and distinct in L II and III. They are scarcely discernible in L I; this is characteristic of auricular flutter. At a later date, while the auricular speed remained the same (300 per minute), the ventricular rate varied from 60 to 100 per minute. There is no group-proportion discoverable in the varying ventricular rates; complete heart block is present. After 10 drachms of tincture of digitalis, given over a period of 10 days, there was no diminution of auricular speed; the ventricular activity was arrhythmic. There was also a difference in the heights of the R deviations. This difference was not of respiratory origin; it may have been due to differing degrees of ventricular excitability from digitalis medication. The electrocardiogram (Fig. 172) was derived from a case of acute endopericarditis without decompensation; flutter developed during the course of acute rheumatic joint manifestations. In this instance, digitalis did not have the effect sometimes found in flutter with cardiac decompensation, namely, first, auricular fibrillation; later, the resumption of normal rhythm with cessation of medication (Chapter XX). As stated, there was no decompensation in this case. The rhythm became normal only when the joint symptoms subsided, the temperature had reached the normal, and the endocardial lesion had become quiescent. From the clinical course, I conclude that the cause of the flutter was the acute endocarditis. In what manner the latter produced the arrhythmia it is impossible to state. One year after the onset of the disease, the rhythm was still normal and the signs of the endocardial lesion (mitral regurgitation) were the same.

**Clinical Recognition of Auricular Flutter.**—In the exceptional instances of this arrhythmia in which the veins of the neck are tremendously distended by each auricular systole, the diagnosis becomes at once evident on inspection and estimation of the rapidity of the jugular pulsations. The later, however, are more often minute and scarcely distinguishable as separate beats.

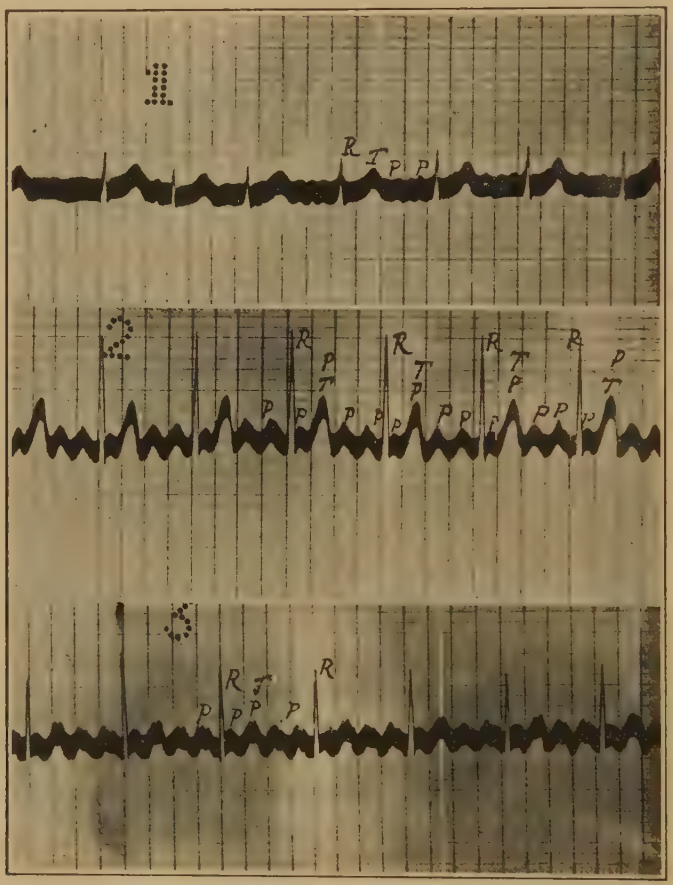


FIG. 172.—Auricular flutter, incomplete heart block, the ratio is 4:1. The auricular rate is 300 per minute; the ventricular, 75. Note that the P waves in L I are almost indistinguishable; they are prominent and contiguous in L II and L III.

Although not thus definable as individual waves, the jugular pulsations are usually sufficiently distinct for one to note that their number greatly exceeds that of the ventricular contractions. In this manner the diagnosis of flutter may occasionally be ventured; but most diagnostic reliance must finally be placed upon recognition of heart block with the varying auriculo-ventricular ratios typical of flutter. If, for example, incomplete heart block at a constant 2:1 ratio is present, the pulse and heart action are regular; the rate, usually between 90 and 120. If the auriculo-ventricular ratios are incon-

stant and constitute changing multiples, as from 2 : 1 to 4 : 1 or the reverse, the diagnosis of flutter can be made from the fact that the ventricular pauses also constitute similar multiples. If the auriculo-ventricular ratio constantly varies, however, or if complete heart block is present, then ventricular action and pulse become arrhythmic and clinically resemble the irregular action of frequent extrasystoles or of auricular fibrillation. Clinical differentiation

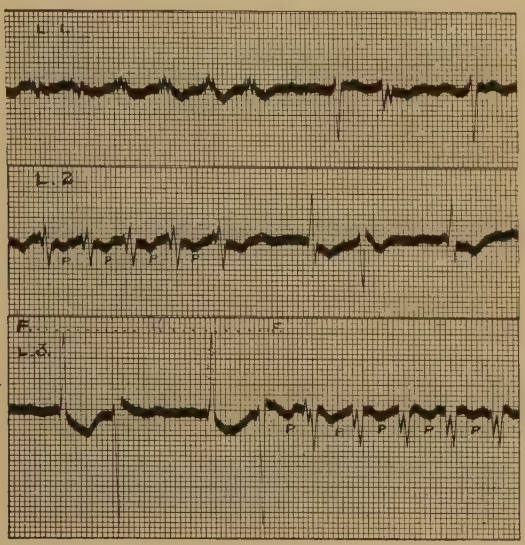



FIG. 173.—Paroxysmal auricular tachycardia. F....F (L III) shows change of paroxysmal tachycardia to auricular fibrillation (absence of regularly recurring auricular complexes). (*Courtesy of A. E. Cohn.*)

of auricular flutter may then be possible from auricular fibrillation by the absence of its characteristic jugular pulse; and from extrasystoles by the absence of compensatory pauses.

### A. III. (3) INCOORDINATION INTERMEDIATE BETWEEN FLUTTER AND FIBRILLATION

In a few instances, I have observed a state of irregular auricular activity which may be regarded, I believe, as an intermediate stage of incoordination between flutter and fibrillation. The auricular rate was usually between 110 and 150; the beats, as shown by the differences in the auricular complexes, came from many scattered ectopic foci; the auricular rhythm was irregular. The ventricular rhythm was also irregular; the rate usually between 100 and 120. Many of the ventricular electrocardiographic complexes varied in the length of their deviations; very few ventricular extrasystoles were present; the picture betokened impulses following many vicarious paths in the ventricle. Although the electrocardiographic picture of the

ventricular arrhythmia was not as distinct as was the case with the irregular (ectopic) auricular activity, it seemed probable that ventricular arrhythmia was likewise due to incoordinate activity, but of a degree considerably less than in ventricular fibrillation (q. v.).

The patients who presented this rare and interesting arrhythmia were in the terminal stages of pneumonia or of cardionephritis. It seemed possible, from the clinical pictures presented, that several factors deserved etiological consideration: *e.g.*, in pneumonia, the toxins; in cardionephritis, retained exerementitious products. In all, there were probably changes in the intracardiac circulation which profoundly affected the nutrition of the general musculature, as well as of the auriculo-ventricular conduction system.

Although, as indicated, irregular auricular activity may be due to two fundamentally different causes (abnormal passage of impulses and auricular incoordination), it is interesting to note the gradations and transitions in auricular irregularity. First are the isolated auricular extrasystoles. These, if numerous and originating outside the sinus area, give rise to auricular tachycardia. The rate is then between 150 and 225. If auricular speed be increased from 225 to 350 per minute, flutter results. This appears to be the limit of regular auricular activity in man. Any further increase in rate results in irregular activity—auricular fibrillation. The fibrillatory waves may be as numerous as 900 per minute. In some instances it is possible to observe a transition from auricular flutter to fibrillation. Between flutter and fibrillation is the intermediate type of inchoate auricular activity just described. There is in addition another transition stage called impure flutter (Fig. 161) that can be diagnosed only from the electrocardiogram. The fibrillary waves show a transient tendency to become as regular as those in flutter, to again break into the irregular waves of auricular fibrillation.

**Cause of Auricular Fibrillation.**—In this connection it is of interest to point out the newest physiological conception of the fundamental cause of auricular fibrillation. Some years ago, the latter was thought to be due to pathological changes (usually sclerotic in nature) in the pacemaker. But some cases that fibrillated during life did not show such changes at autopsy. It was also assumed that auricular fibrillation was caused by frequent abnormal excitation originating in or outside the sinus area. The actual explanation for auricular fibrillation seems to lie in the fundamental observations of Garrey and Mines. Working independently, they showed that if the auricles are made to fibrillate by faradization, and if then a portion of the auricular tissue (*e.g.*, the auricular appendix) is clamped off, fibrillation will continue in the remainder of the auricular musculature. With removal of the clamp, the entire auricle again fibrillates. They also found that if the auricle is incised trouser-fashion so that sufficiently broad auricular strips remain connected by sufficiently broad auricular bridges, and then the auricle be faradized anywhere, contraction of the auricular strips will continue long after faradization has ceased. Garrey calls such contractions "circus con-

tractions" for he assumes that such an excitation wave travels in a continuous circuit, and that the impulse spreads from fiber to fiber. In the experimentally slit auricle, the excitation impulses are irregularly and sinuously blocked because of the difference in refractory periods of the incised fiber groups and because impulse conduction is interfered with. Thus while the excitation continues its "circus" path, the auricular strips are actually contracting at various times and with varying energies. The result is the incoordinate, auricular tremor which we denote clinically as auricular fibrillation. Lewis and his coworkers have recently corroborated Garrey's observations in a series of experimental and clinical papers. Lewis has likewise shown that auricular flutter is due to the breaking up of the excitation wave in a series of sinuous movements. He regards flutter (especially impure flutter), as a gradation of auricular fibrillation.

Concisely stated, therefore, auricular fibrillation is essentially due to any influence—toxic, nutritional, reflex etc.—which breaks up the simple, normal, single, "circus" wave starting from the pacemaker by interfering with and blocking the propagation of the wave.

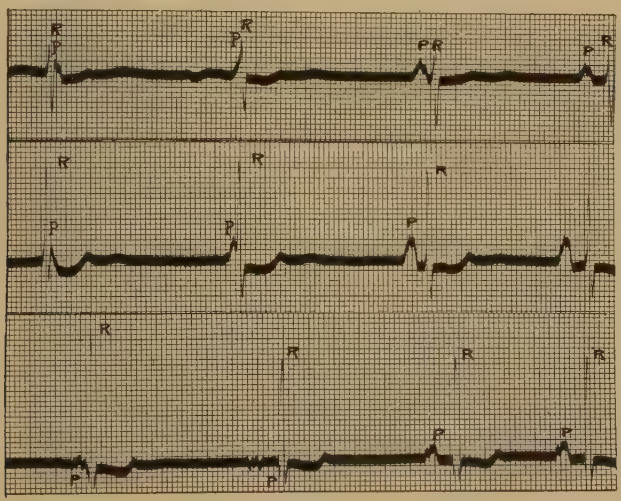


FIG. 174.—Nodal extrasystoles showing coincidence of P and R waves. (*Courtesy of A. E. Cohn.*)

### A. NODAL EXTRASYSTOLES

Premature contractions having their origin in the auriculo-ventricular node are called nodal extrasystoles. The auricular and ventricular impulses start simultaneously, or almost so from their nodal origin. Hence, in the jugular tracings, the *a* and *c* waves, and, in the electrocardiogram, the P and R waves fall coincidentally (Fig. 174). In the phlebrogram, a large summation *a-c* wave is produced (Figs. 175, 176); in the electrocardiogram,

the small P is often lost in the larger R complex. This type of extrasystole cannot be diagnosed without the use of graphic methods.

### B. VENTRICULAR ARRHYTHMIAS

**1. Ventricular Extrasystoles.**—As with the auricular type, ventricular extrasystoles depend upon the abnormal passage of the impulse through the ventricle.

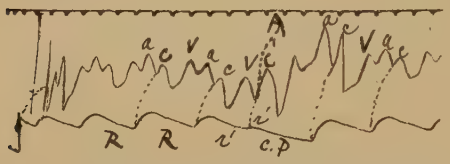



FIG. 175.—Nodal extrasystole, graphically illustrated by the synchronous occurrence of the foot points of A'c'.

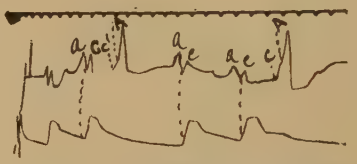



FIG. 176.—Nodal extrasystole which has not produced a pulse wave. (*Frustrane contraction.*)

Among other distinctions between such aberrant (heterogenetic) ventricular beats (ventricular extrasystoles) and those arising in the auriculo-ventricular node—true idioventricular rhythm—is the one of rate. The node originates rhythmical contractions at a speed of from 25 to 40 per minute (heart block). The rate of extrasystolic ventricular rhythm is between 130 and 200 per minute.

In ventricular extrasystoles (Fig. 112) the auricle follows its normal rhythm and contracts when the ventricle is in a refractory state; and, as a

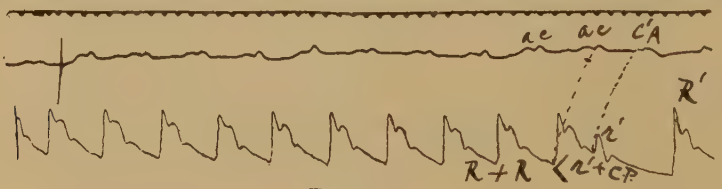



FIG. 177.

FIG. 178.

FIGS. 177, 178.—The ventricular extrasystole plus the compensatory pause is slightly greater than two rhythmic beats. (Increased compensatory pause, C.P.).

result, there is no ventricular response until the next succeeding ventricular contraction. Thus the time of ventricular prematurity, added to the compensatory pause, equals two rhythmic beats. This fact is of value in the recognition of ventricular extrasystoles from radial tracings alone, or in polygraphic tracings in which the jugular is not sufficiently clear to be of value.

In auricular extrasystoles, both chambers contract prematurely, and the succeeding pause may or may not be compensatory (Fig. 120). Ventricular extrasystoles, however, are sometimes followed by pauses which are not exactly compensatory in length (Figs. 177–180). Occasionally extrasystoles

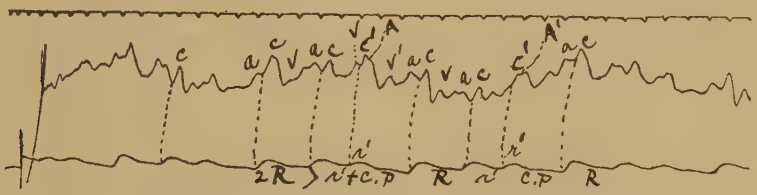



FIG. 179.—Ventricular extrasystole plus compensatory pause is less than two rhythmic beats (diminished compensatory pause (C.P.).

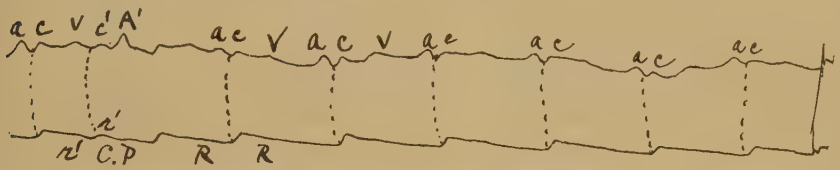



FIG. 180.—Ventricular extrasystole without compensatory pause (C.P.).

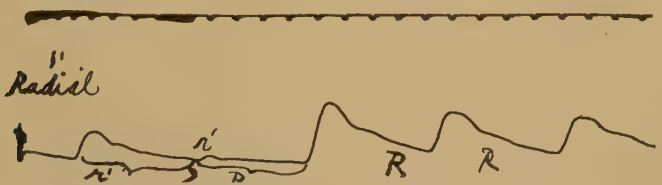



FIG. 181.—Extrasystole which is not "premature." r′ is greater than P (the pause following the extrasystole).

occur so late that they lose their characteristic "prematurity" (Fig. 181). The amplitude of the radial beat following the extrasystolic pause in any type of premature contraction is usually larger than that of the normal beat, and expresses graphically its greater strength (Figs. 112, 114, 181). This is sometimes regarded as indicative of favorable cardiac recuperative power.

### B. II. INTERPOLATED VENTRICULAR EXTRASYSTOLES

When the cardiac rate is slow, ventricular extrasystoles do not always interfere with the time of the next normal ventricular contraction; these are known as "interpolated" extrasystoles. This type of premature contraction occurs in the course of normal rhythm, and is not followed by a compensatory pause. Such extrasystoles are found in mid-diastole and in groups, rarely as isolated phenomena. Figure 88 is therefore of interest because it shows a single interpolated extrasystole (L II Ex.). The electrocardiogram was derived from a male patient 54 years old who was extremely decompensated. He had had syphilis 30 years previously. The Wassermann blood reaction

was positive. He had severe myocarditis and marked left ventricular hypertrophy. Fluoroscopy and radiography demonstrated aneurismal dilatation of the entire thoracic aorta. Under vigorous antiluetic, and the usual treat-

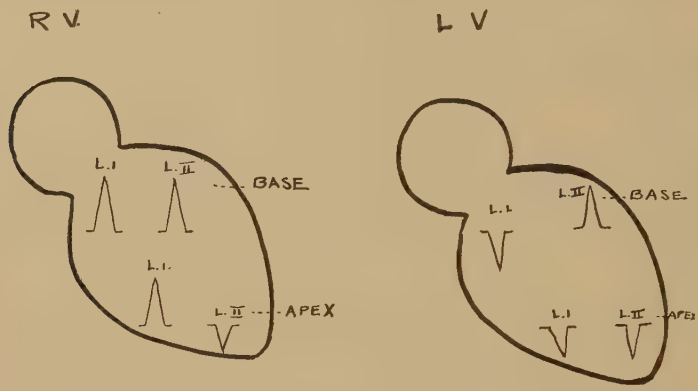



FIG. 182.—Schematic view of types of ventricular extrasystoles arising from various ectopic foci, showing the direction of the corresponding R deviations. R.V., right ventricle. L.V., left ventricle.

ment for decompensation, the patient improved to such an extent that at the last examination he considered himself well. The electrocardiogram is of further interest because it gives some corroboration of the clinical findings: Negative T in L I and an abnormally wide R deviation in L I and III,

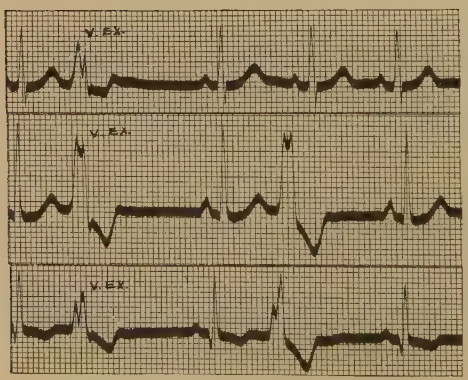



FIG. 183.—Ventricular extrasystole (V.Ex.) from right ventricle near the base. (*Courtesy of A. E. Cohn.*)

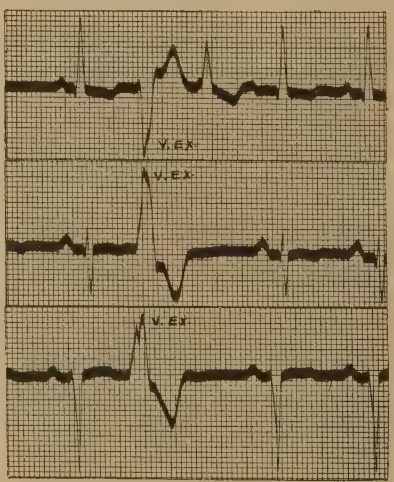



FIG. 184.—Ventricular extrasystole (V.Ex.) from left ventricle near the base. (*Courtesy of A. E. Cohn.*)

presumed evidences of myocarditis (Chapters IX, XXXII); the positive R I and negative R II and R III also confirm the clinical and orthodiascopic findings of a markedly hypertrophic left ventricle.

Electrocardiograms of ventricular extrasystoles vary considerably from normal complexes because their course in the heart (Fig. 70, J. K.) is quite different from that followed by the normal excitation wave. The extrasystoles may originate in either ventricle. There is experimental evidence that extrasystoles arising from the various areas of the ventricular musculature—so-called ectopic foci (Lewis)—conform to definite electrocardiographic types. By analogy, it is possible to determine in the human being the abnormally excited areas in the ventricles from variations in the form of the atypical electrocardiograms (Fig. 182). However, one must remember that the abnormal path followed by extrasystoles need not necessarily indicate a corresponding abnormal ventricular focus as its starting point.

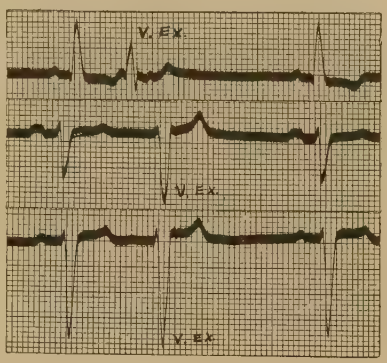



FIG. 185.—Ventricular extrasystole (V.Ex.) from the left ventricle near the apex. (*Courtesy of A. E. Cohn.*)

Although studies of the various forms of ventricular extrasystoles are of considerable interest and importance, there is at present no correlation, except in isolated cases, between these different types and the clinical condition. It is to be noted that the P deviation is usually not visible because it is lost in the larger ventricular complex; occasionally, however, it appears at such time that it distinctly notches the R wave.

A schematic view of the usual aberrant foci and their corresponding electrocardiographic types is given in Fig. 182; it shows the direction of the R and T deviations in the L I and II. These types are also illustrated by examples of ventricular extrasystoles derived from human beings (Figs. 183, 184, 185). That there are intermediate types coming from various intermediate aberrant foci one may assume from certain rare cases of ventricular extrasystoles with heart block showing gradual and regularly varying abnormal complexes (Fig. 186). It is as if the abnormal excitation had affected, ladderwise and in progression, various successive areas of the ventricular musculature.

Instances of regularly recurring extrasystoles (coupled rhythm) are given in Figs. 187, 188–A, 188–B. Occasionally several originate in the same ectopic focus (Fig. 189), producing short runs of paroxysmal tachycardia of ventricular origin.

### B. III. AUTOMATIC VENTRICULAR ACTIVITY—VENTRICULAR ESCAPE

This rare type consists in the sudden "escape" of the ventricle from auricular control, and results in ventricular automatism or independent activity. In a few reported cases, there was marked retardation of the auricular, and,

in most instances, of the ventricular rate. In the case that I observed there was never any marked pulse retardation (Fig. 190); the lowest rate was 55 per minute, and the auricular speed was not abnormally slow. Its etiology will be discussed in a later chapter (Chapter XI).

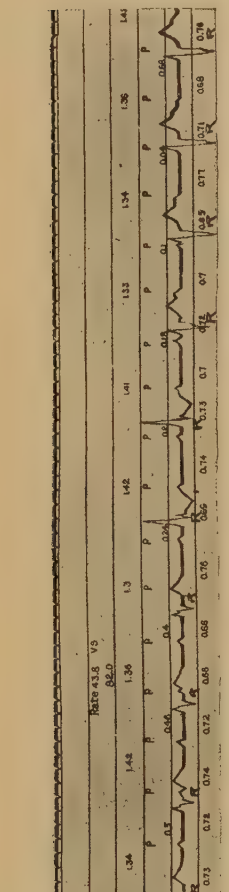


FIG. 186.—Extrasystoles arising from various ventricular foci, as shown by the differences in R complexes. Complete heart block present. (*Courtesy of A. E. Cohn.*)

When auricles and ventricles beat at such rates that their waves and deflections in the tracings are regularly superimposed, their origin in or near the auriculo-ventricular node is assumed; these are called nodal extrasystoles (q. v.). Such simultaneous action is seen in parts of the electrocardiogram of the case of "ventricular escape" (Fig. 191 S); however it is apparently due to temporarily identical auricular and ventricular speeds, for as these vary, varying *a-c* and *c-a* (Fig. 190) or P-R and R-P intervals (Fig. 191) soon occur. Besides, nodal beats are usually either regularly interpolated in the normal rhythm, or when premature, are followed after longer or shorter compensatory pauses by the normal dominant beat.

The assumption of nodal extrasystoles is therefore not warranted, I believe, by the electrocardiogram of the case of ventricular escape here shown (Fig. 191), all of whose complexes are alike. Backward conduction (q. v.) from ventricle to auricle, a rare reversal of the cardiac mechanism, also requires consideration as a possible explanation. In a case of backward conduction reported, that rhythm, when established, showed a definite ventriculo-auricular conduction time similar to the normal, and was accompanied by marked ventricular slowing. This conception if applied to my case would necessarily assume that the "reversed mechanism" suddenly and irregularly ceased from time to time in those parts of the tracing which do not show a "retrograde conduction time" (*c-a* or R-P intervals) of less than one fifth second, and that frequently, isolated beats were retrograde. This hypothesis is highly improbable and is not supported by the electrocardiogram. Rihl describes a case of occasional automatic ventricular action produced by vagal pressure. Lewis reports a case of rheumatic mitral stenosis with decompensation; digitalis had been given with resulting ventricular automaticity ("ventricular escape," Lewis). Gallavardin, Dufourt, and Petzetakia describe three cases with slow pulses (in one case the rate was 36 per minute) in which there was no clinical evidence of organic cardiovascular disease. In two,

numerous polygraphic and electrocardiographic tracings showed the occurrence of ventricular automatism; in all three, it was readily evoked by ocular and vagus pressure, and by atropin injection. The writers suggest two causes for the phenomena: Relative retardation of the auricular as com-

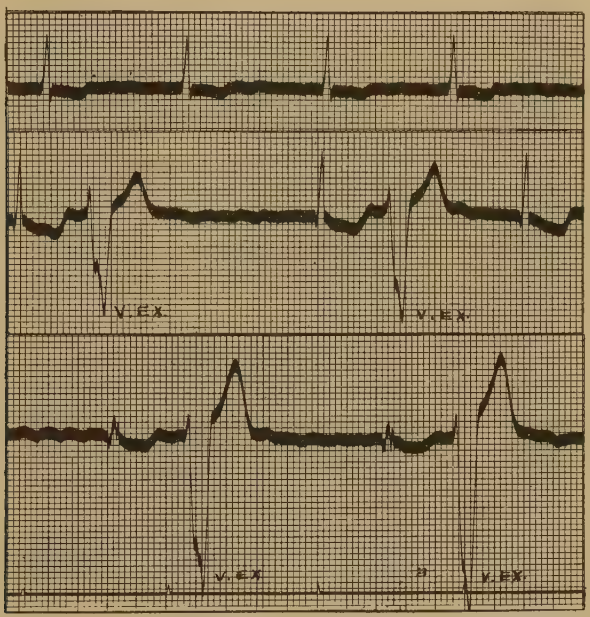



FIG. 187.—Regulary recurring ventricular extrasystoles (V.Ex.); coupled rhythm (pulsus bigeminus). (*Courtesy of A. E. Cohn.*)

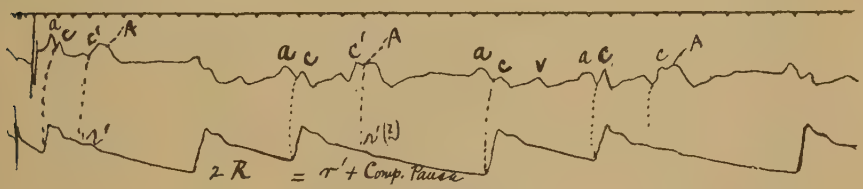



FIG. 188*A*.

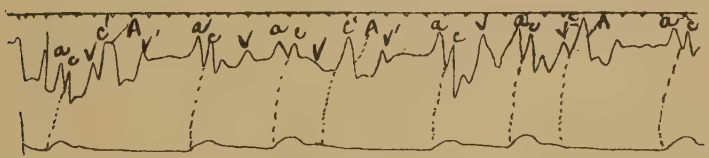


FIG. 188*B*.

FIGS. 188A and 188B.—Frustrane ventricular extrasystoles; coupled rhythm.

pared with the idio-ventricular rate, that is, the ventricular rate is increased when the ventricle beats independently of auricular control; or acceleration of the ventricles beyond the auricular rate. Two of their cases had very slow auricular rates occurring either spontaneously or induced by the methods

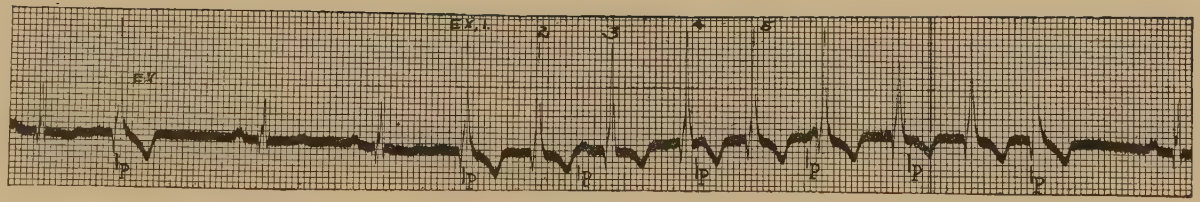


FIG. 189.—Short runs of paroxysmal tachycardia of ventricular origin (V.Ex. 1, 2, 3, 4, 5, etc.). The normal beats are marked P-R-T.

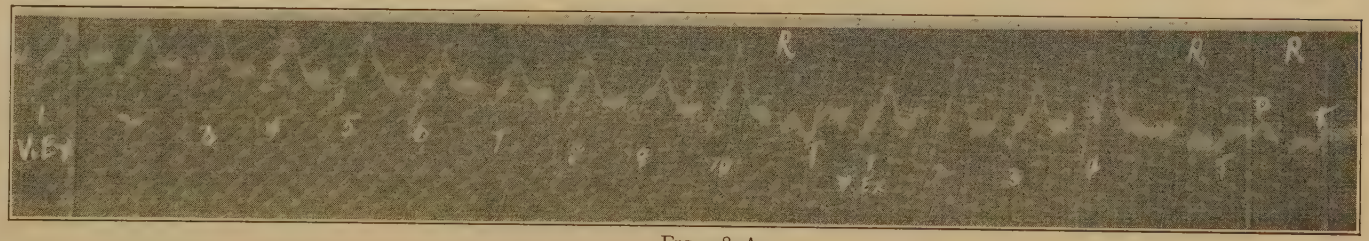


FIG. 189A.

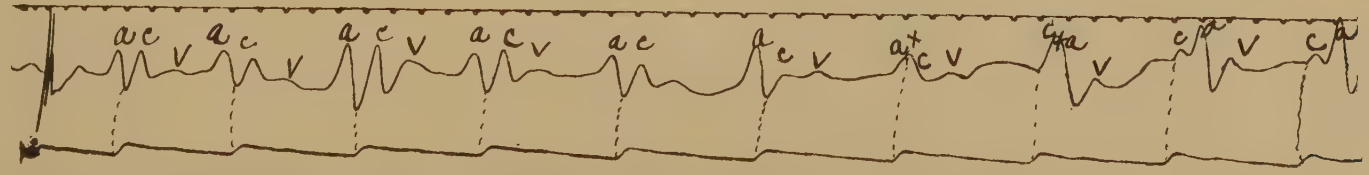


FIG. 190.—Ventricular escape. Part of a long series of tracings showing at times normal rhythm; at others, ventricular automatic activity (ventricular escape). Retrograde conduction from ventricle to auricle (backward conduction) could be excluded because of the irregular *c-a* intervals.

described; the third showed no auricular retardation on vagus or ocular pressure, or after atropin injection. The arrhythmia in the first cases was apparently due to relatively increased idioventricular (auriculo-ventricular nodal) rapidity beyond that of the sinus node. Except for a very slight change in the complexes of the automatic ventricular beats in two of the cases—the absence of a very small S wave—all of the complexes were identical. In digi-

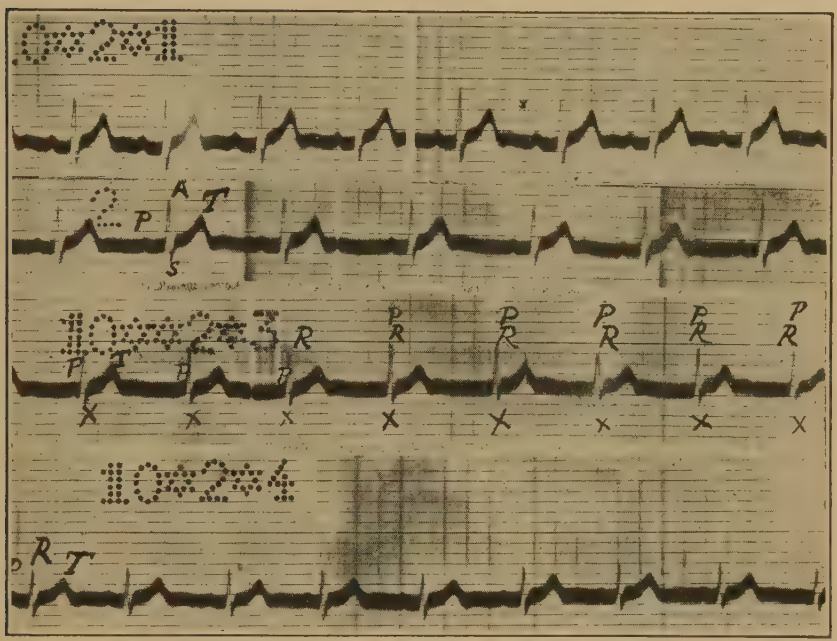



FIG. 191.—Ventricular escape. Sections of a continuous tracing. At X the ventricle "escapes" from auricular control and beats independently at such rate that auricle and ventricle occasionally contract synchronously (superposition of P and R waves). The rhythm is normal in the other parts of the tracing.

talis poisoning, Cohn and Fraser have occasionally found either identical auricular and ventricular speeds, or ventricles beating more rapidly than auricles, with ventricular escape. In the case I here report there was at no time any marked pulse retardation—the lowest rate was 55—nor was there any evidence of auricular slowing, although there was, at the periods of ventricular automatism, some slight difference between auricular and ventricular rapidity. Except for occasional somewhat slower beats, the idioventricular and normal ventricular rates were approximately the same. Slight sinus arrhythmia was sometimes present, but was not more marked than is frequently found as a physiological phenomenon.

### B. IV. PAROXYSMAL TACHYCARDIA OF VENTRICULAR ORIGIN

As with the auricular, paroxysmal tachycardia of ventricular origin is marked by a rapid succession of ventricular extrasystoles (Fig. 189). It

Fig. 192.—Ventricular fibrillation. (*After Robinson and Bredeck.*)

differs from auricular paroxysmal tachycardia (auricular tachystole) in that the latter originates a new rhythm by starting in an auricular focus outside of the normal pacemaker, while paroxysmal ventricular tachycardia consists of a sudden run or shower of ventricular extrasystoles. The diagnosis can naturally be made at once from the electrocardiogram (Fig. 189) by the difference in form of the extrasystolic beats. A paroxysm usually comprises from 8 to 10 premature contractions; very exceptionally, there may be as many as 25 or 30. Sometimes there are several runs of rapidly recurring paroxysms that make up an entire attack, so that to the patient, it may seem one long-continued paroxysm.

Clinically, paroxysmal ventricular tachycardia can be distinguished from the auricular variety by its shorter course; by the pulse deficit, for many of the extrasystoles do not produce pulse waves; by the strong "thumps" heard at the apex indicating the extrasystoles; by the absence of regular rapidly recurring auricular (*a*) waves in the neck; and finally by the fact that ventricular tachycardia is not controlled by pressure of the vagus in the neck.

### B. V. VENTRICULAR INCOORDINATION

1. **Ventricular Fibrillation,** similar to the incoordination of auricular fibrillation, is due to incoordinate ventricular activity. It has only been very rarely studied electrocardiographically in the human being (Fig. 192). When it occurs, it is usually followed by death within a very few minutes. The reason for this is that the inchoate ventricular contractions have no propulsive effect upon the blood in the ventricular chambers, and hence blood is improperly or not at all supplied to the systemic circulation. However one case has been reported in which the patient lived 30 hours after an attack of ventricular fibrillation lasting 15 minutes. It is assumed that ventricular fibrillation is sometimes the cause of sudden death in some cases of cardiac decompensation, in which at autopsy no other cause can be found.

**2. Bundle-branch Lesions.**—The simplest type of ventricular incoordination consists of asynchronous ventricular contraction from lesions of the main branches of the auriculo-ventricular bundle. Such lesions may be permanent or temporary. In bundle-branch lesions, the impulses originate in the A-V

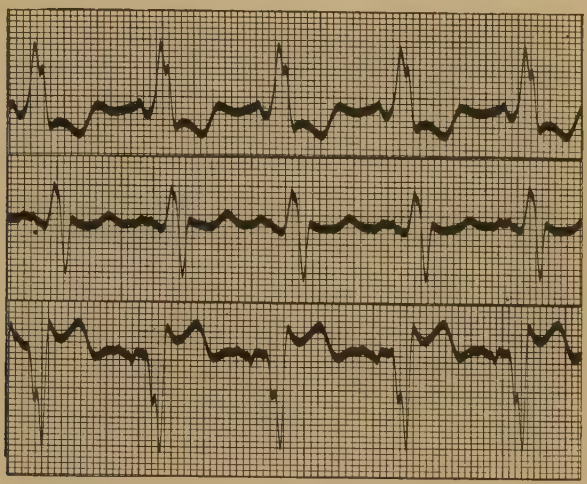


FIG. 193.—Lesion of the right branch of the conduction system. Conduction takes place along the left branch. The initial deflections are tall and positive in L I, and tall and negative in L III. (*Courtesy of A. E. Cohn.*)

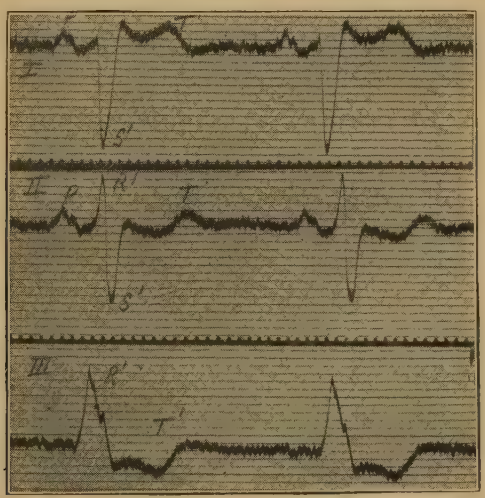



FIG. 194.—Lesion of the left branch of the conduction system. Conduction takes place along the right branch of the bundle. The initial deflections are tall and positive in L III, and tall and negative in L I. (*After E. P. Carter.*)

bundle (junctional tissue) before its bifurcation, and follow the unblocked branch in the ventricle (Fig. 70, H.I.). Experimental and clinical observations have shown that bundle-branch lesions produce characteristic

electrocardiograms. Microscopic sections of post-mortem specimens have corroborated and confirmed the electrocardiographic evidence in a few instances.

Since the excitation wave travels along the healthy branch and excites the corresponding ventricle to contraction, the electrocardiogram in the various leads shows the characteristics of contraction of one ventricle alone, without the counterbalanced contraction of the other. Hence for example, in lesions of the right branch, by far the more common, the initial deflections show left-sided conduction. The deviations are tall and positive in L I, and deep and negative in L III (Fig. 193). The opposite is the case in the left-sided lesions (right-sided conduction) (Fig. 194). A further characteristic of bundle lesions is the abnormal length of time required for the completion of the excitation wave, the width of the R usually being about 0.10 second. In addition, the deviations are frequently notched, the ventricular complex usually diphasic. The T wave is commonly deviated in a direction opposite to that of its accompanying R peak.

The electrocardiograms of cardiac hypertrophy are somewhat similar to those of branch lesions. They are differentiated by a narrower R, the lesser amplitude of their deflections, the direction of the T which is the same as that of the R, and by the tri- or quadriphasic character of the ventricular complexes.

The cardiac rate is normal in bundle branch lesions. The diagnosis can occasionally be made by hearing a definite split first sound at apex, probably due to the asynchronous ventricular activity present in such cases.

### C. TRUE BRADYCARDIA

This term should be applied only to types of slow, regular heart action in which there is normal conduction time from auricle to ventricle and in which each beat represents a sequential auriculo-ventricular contraction. Thus defined, it has its proper place in a classification of arrhythmias. Otherwise, when loosely used in a sense of slow pulse rate (so-called spurious bradycardia), it takes no cognizance of auricular rhythm or of auriculo-ventricular conduction, and may therefore haphazardly include such divergent cardiac irregularities as auricular fibrillation, extrasystoles, and heart block (q. v.). It is difficult to fix a definite rate as typical of true bradycardia. Cardiac and pulse rates ranging from 60 to 65 per minute are by no means rare in normal healthy adults, especially in those with rather marked vasomotor instability, flushed hands and face, sudden irregular pallor, and cold and moist extremities. Examples of regular, slow heart action due to extracardial causes are given in Figs. 195–199. An instance due to salicylate of soda is shown in Fig. 198.

In the electrocardiogram of true bradycardia, all the ventricular complexes are alike; there is slow, regular heart action. Thus, Fig. 199 taken

from a young epileptic with a normal cardiovascular apparatus shows regular sequential heart beats at the rate of 50 per minute.

**Clinical Recognition of True Bradycardia.**—The rate is rarely below 40 per minute; the cardiovascular apparatus is often organically normal.

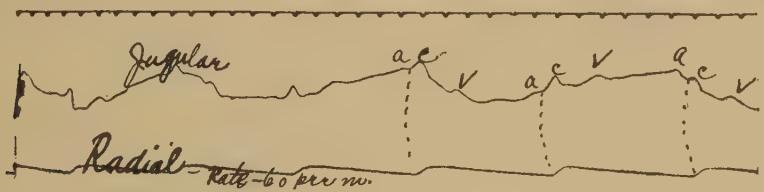



FIG. 195.

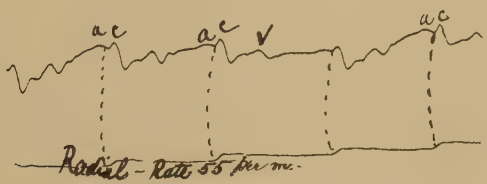



FIG. 196.

FIG. 197.

FIGS. 195–197.—True bradycardia from patients with gastric symptoms and normal hearts.

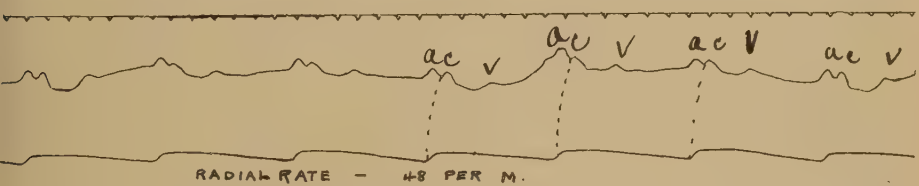



FIG. 198.—True bradycardia caused by salicylate of soda.

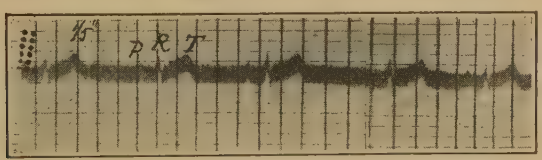


FIG. 199.—L I, True bradycardia. The auricles and ventricles beat sequentially. The rate is 50 per minute.

Since in true bradycardia the auricles and ventricles beat sequentially, the jugular waves (*a* waves) are seen to precede the carotid pulsations (*c* waves). True bradycardia must be differentiated from the slow heart action found in heart block. In the former, the jugular pulsations occur with the same frequency as the carotid, the ventricular, and the pulse beats; in

heart block, jugular pulsations occur more frequently than the carotid. Jugular pulsations may be rendered more prominent by having the patient lay flat and then inspecting the right side of the neck. By holding a white card in the space between the jugular and the carotid, it is sometimes possible to reflect the jugular pulsations on the card by placing the patient in a proper light.

### D. ARRHYTHMIAS PRODUCED BY ABNORMAL SEQUENCE OF CONTRACTION OF AURICLES AND VENTRICLES

This sequence may be disturbed either at the pacemaking area (the sinoauricular node) or at the junctional tissues (the atrio-ventricular node).

**D I (1) Sinus Arrhythmia.**—This pulse irregularity is produced both by physiological and abnormal influences which affect the sinus region, the pacemaker of the heart. Sinus arrhythmia of the physiological respiratory type consists of alternate moderate acceleration and retardation, a waxing and waning of the pulse rate, corresponding to expiration and inspiration respectively. The arrhythmia is of vagal origin and is ascribed to difference in the vagal inhibitory tone from phasic changes in respiration. It is a physiological phenomenon in children (Figs. 200, 201) and young adults (Fig. 202) ("the youthful irregularity" of Mackenzie), although it is by no means uncommon as a normal variation in the middle-aged, especially upon forced deep respiration. Sinus arrhythmia due to other causes is also illustrated (Figs. 203–206).

In the electrocardiogram, sinus arrhythmia is recognized by varying lengths of the pauses between the beats (Fig. 207); there is no difference in any of the complexes unless phasic respiratory variations (Chapter IX) are present. In such cases there is a gradation in the height of the ventricular complex due to changes in the position of the heart from movement of the diaphragm during respiration.

**Clinical Recognition of Sinus Arrhythmia.**—The physiological type with normal pulse rate is readily diagnosed because it wanes and waxes with inspiration and expiration, respectively. The pathological types of sinus arrhythmia do not usually show this correlation. Their clinical diagnosis is then made by the comparatively slow pulse rate with irregularly long diastolic pauses. The interventricular interval may be sufficiently long to block out an entire auriculo-ventricular contraction, thus producing sino-auricular block (q. v.). The distinction between sinus arrhythmia of the non-respiratory type and auricular fibrillation with slow and fairly regular ventricular activity depends upon the observation of the jugular pulsations. In the former, there are regularly recurring jugular waves (*a* waves) which precede the carotid pulsations; in the latter this relationship naturally does not exist since auricular contractions are absent.

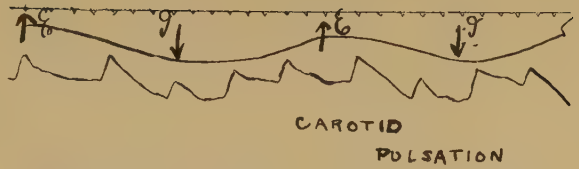



FIG. 200.—Carotid pulse of a normal child, the breathing curve with the inspiratory (I) and expiratory (E) phases, and corresponding difference in pulse rate are shown.

FIG. 201.—Moderate sinus arrhythmia (from a normal child).

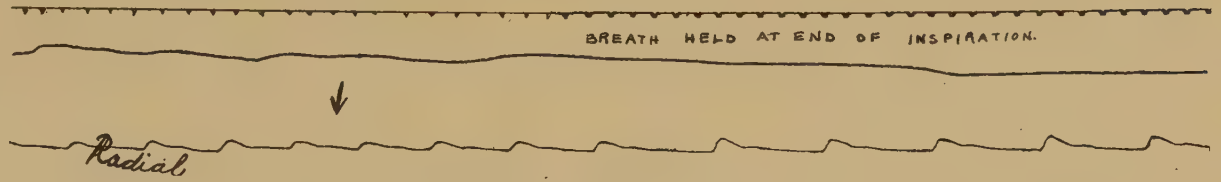



FIG. 202.—Retardation (sinus slowing) from holding the breath at the end of inspiration. The arrow indicates the point at which normal breathing ceased (from a young healthy adult).

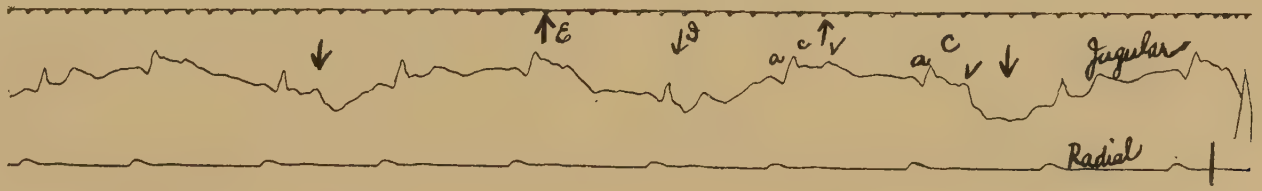



FIG. 203.—Slight sinus arrhythmia at the time of crisis in recovery from pneumonia. I, Inspiration; E, expiration.

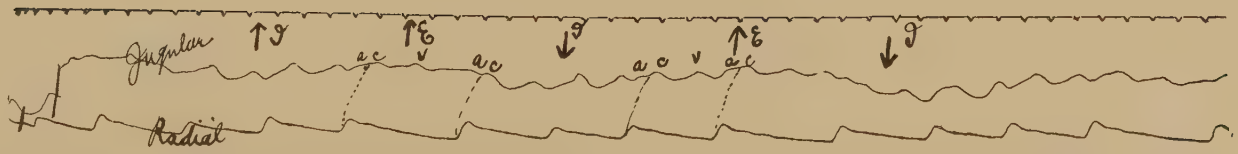



FIG. 204.— Sinus arrhythmia during critical defervescence in a child recovering from tonsillitis.

FIG. 205.—Respiratory sinus arrhythmia in a boy of 15 with an aortic lesion.

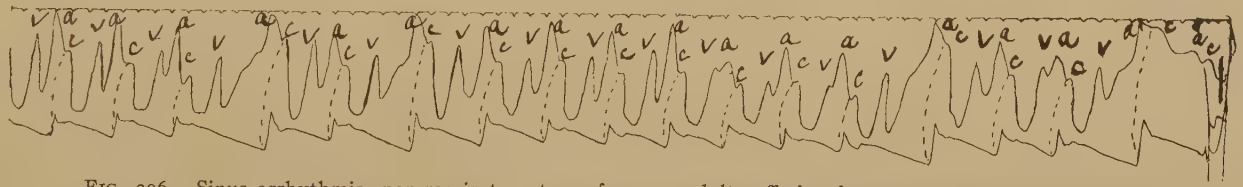


FIG. 206.—Sinus arrhythmia, non-respiratory type, from an adult suffering from severe intestinal hemorrhage.

### D. I. (2) SINO-AURICULAR BLOCK

Occasionally abnormal influences affect the vagus or its endings in the sino-auricular node so that an entire beat is blocked out. This arrhythmia is rare. There is stoppage of the entire heart (Figs. 208, 209). The pause is

FIG. 207.—Electrocardiogram showing sinus arrhythmia of respiratory origin. Note the varying lengths of the diastolic pauses. (*After T. Stuart Hart.*)

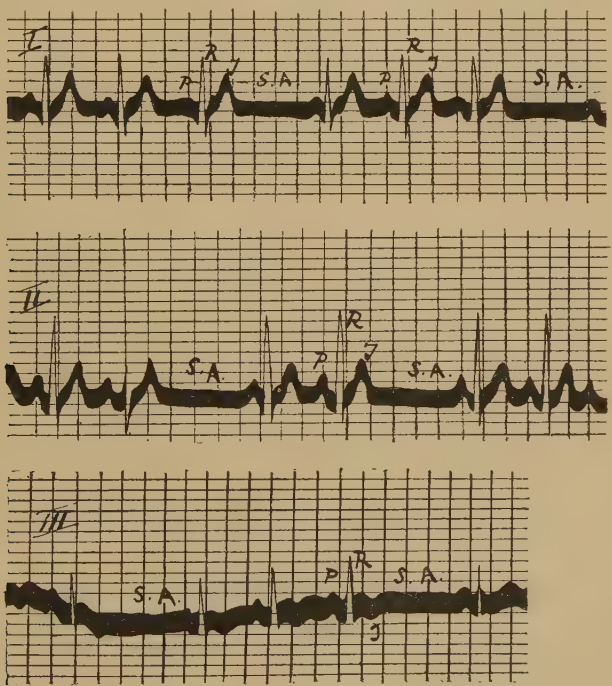



FIG. 208.—Electrocardiogram showing sino-auricular block (S.A.), due to smoking.

usually somewhat less than that required for two normal contractions; when extreme, the pause may represent the time required for three, or even four normal beats. I have seen two cases due to tabagism, one of whom had an organically normal heart; the other patient had myocarditis with mild decompensatory symptoms. Figure 208 is taken from one of these patients.

In addition to the sino-auricular block there is moderate tachycardia. This double effect of nicotine—moderate tachycardia and sino-auricular block—was probably due to the varying action of the poison upon the ganglionic terminations of the vagus and sympathetic nervous system (Fig. 210). In

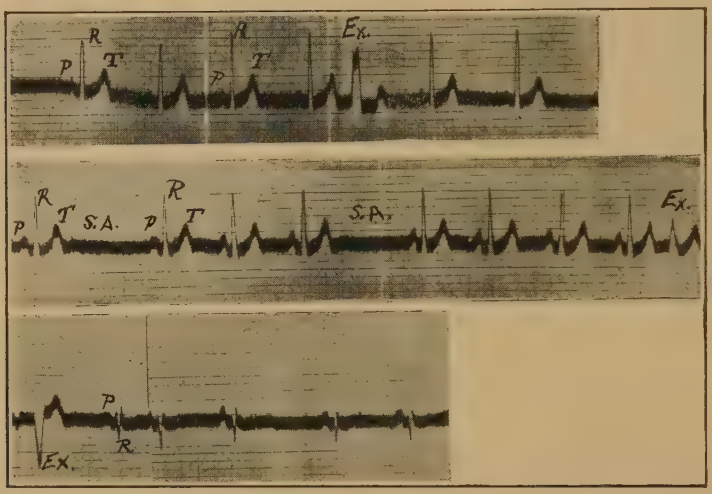



Fig. 209.—Electrocardiogram showing sino-auricular block (S.A.) due to smoking.

the other case, a smoker with myocarditis and decompensation, ventricular extrasystoles (Fig. 209) were also present; sino-auricular block disappeared two days after smoking was stopped; the extrasystoles ceased some days later when compensation was restored by the use of digitalis and theobromin sodium salicylate.

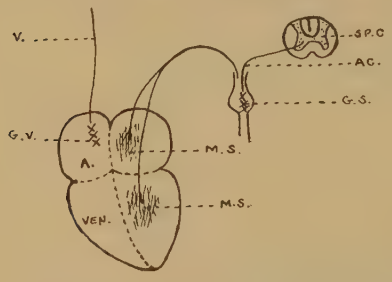



Fig. 210.—Diagram showing the effect of nicotine upon the ganglionic terminations of the vagus and accelerators. (Modified from Cushny) V, vagus; A.C., accelerator fibers; Ven, ventricle; A, auricle; M.S., cardiac musculature; G.V., ganglia at vagus termination; G.S., ganglia at termination of sympathetic nerve; SP.C., spinal cord.

### D. I. (3) BLOCKED AURICULAR BEAT

This is a very rare arrhythmia. It is usually due to digitalis medication. Ventricular action does not follow the blocked auricular impulse, consequently there is a quiescent period equivalent to two beats (Fig. 211). It is distinguished from extrasystoles by the absence of the characteristic small pulse wave and of the extrasystolic heart sounds; from sino-auricular block, only by recognition of an auricular jugular wave in the former and its absence in the latter.

### D. II. (1) PROLONGED CONDUCTION TIME

This represents the simplest type of heart block. Various factors cause moderate prolongation of the impulse from auricle to ventricle, that is, of the P-R time. These are chiefly digitalis medication, myocarditis and acute endocarditis. I have also observed lengthened conduction in several

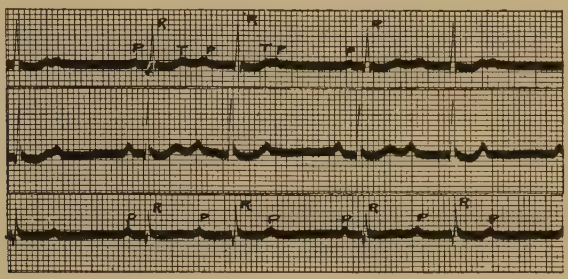


FIG. 211.—Blocked auricular beat showing absence of ventricular response, as well as prolonged conduction time. (*Courtesy of A.E. Cohn*).

patients with auricular extrasystoles of functional origin. Cases of extreme prolongation of the P-R time up to or even more than 0.50 of a second sometimes occur. For example, Fig. 212 is the electrocardiogram of a male patient, aged 62, who had myocarditis and nephritis; at the time that the

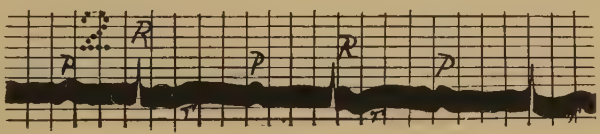


FIG. 212.—Electrocardiogram showing very slow ventricular rate (37 per minute) with a P-R time of 0.8 second. (From a patient with severe myocarditis.)

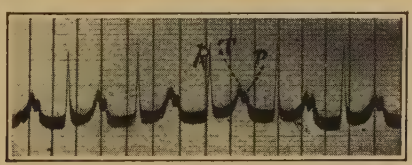


FIG. 213.—Prolonged conduction time. P-R, 0.3 second (from a patient with acute rheumatic endocarditis).

electrocardiogram was taken he was suffering from a third attack of severe decompensation: dyspnoea, oedema of the legs and ascites. He died later with uremic symptoms. The electrocardiogram shows a ventricular rate of 37 per minute with a conduction time of 0.8 second. A less marked instance is shown in Fig. 213. Prolonged conduction time can only rarely be recognized clinically by observing the prolonged interval between the *a* and *c* waves in the neck.

### D. II. (2) SHORTENED CONDUCTION TIME

The normal P-R interval is from 0.16 to 0.20 second. This interval—the conduction time from auricle to ventricle—is occasionally shortened in both paroxysmal and simple tachycardia; however, as already stated, rapid cardiac activity in these arrhythmias occurs mainly at the expense of diastole.

### D. II. (3) BACKWARD CONDUCTION FROM VENTRICLE TO AURICLE

This exceedingly rare anomaly is exemplified in Fig. 214, a case reported by Williams and James. It shows the auricular (P), regularly *following* the ventricular beat. Backward conduction was also corroborated in that case by fluoroscopic examination. The patient, a laborer of 51, suffering from diarrhea for one year, had attacks of Stokes-Adams syndrome. The cardiovascular system was apparently normal organically. The arrhythmia persisted for many months and was not affected by atropin injections. Finally, the rhythm again became normal. The writers suggest as possible etiological factors abnormal vagus control or a toxic cause.

### D. II. (4) (a) INCOMPLETE HEART BLOCK

In its simplest type, this consists in the absence of ventricular response to auricular impulses. There is a geometric ratio between auricular and ventricular beats; for example, the ventricle responds to every second, third or fourth auricular impulse. Such cases are termed incomplete heart block at a 2:1, 3:1, etc. ratio respectively (Figs. 215, 216). The ventricle and pulse beat rhythmically unless disturbed by occasional premature ventricular contractions (Fig. 216, *c*). In other types of incomplete block the ventricular response varies irregularly from one ratio to another; the ventricle, for example, answers haphazardly every second or third or fourth auricular impulse. Thus the pulse becomes correspondingly irregular. In both incomplete and complete block, the auricles beat rhythmically at approximately normal speeds, 60 to 80 times per minute. In incomplete heart block, the ventricular rate is considerably diminished; as stated, its regularity depends upon the auriculo-ventricular ratio. The clinical recognition of a 2:1 heart block lies in noting that the jugular (*a*) pulsations are twice as numerous as the *c* waves. In addition the auricular beats may be occasionally heard as fainter heart sounds interspersed between the ventricular beats.

### D. II. (4) (b) COMPLETE HEART BLOCK

This is said to exist when there is no relationship between auricular and ventricular speeds. The ventricle follows its own inherent independent rhythm (Figs. 217–226). This is called the idioventricular rate, the rate initiated by the atrioventricular node, and is usually between 25 and 40

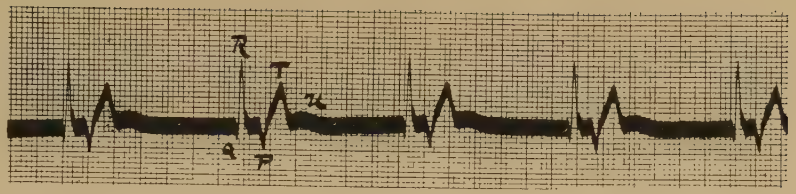
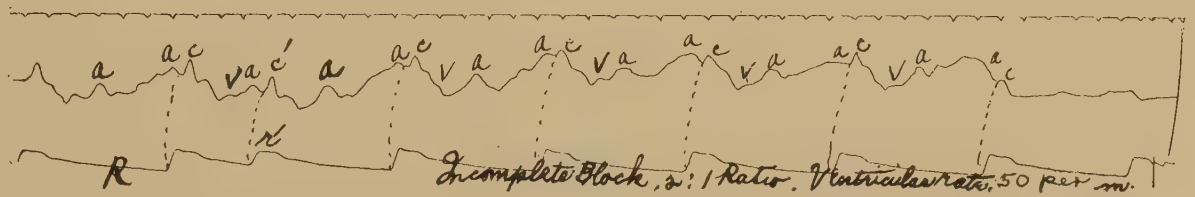



FIG. 214.—Electrocardiogram showing backward conduction from ventricle to auricle. (*After William and James.*)

FIG. 215.




FIGS. 215, 216.—Incomplete block with a 2:1 ratio. In Fig. 216 there is a premature ventricular contraction (*c'*). The preceding auricular beat is not premature. The pause following *r'* is not compensatory but is equal to an entire rhythmic beat.

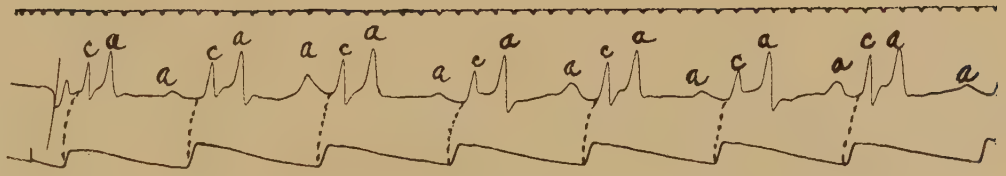


FIG. 217.

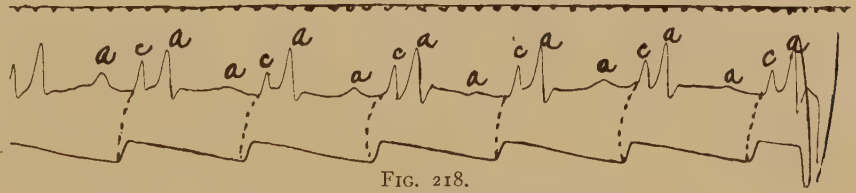


FIG. 218.

FIGS. 217, 218.—Complete heart block.

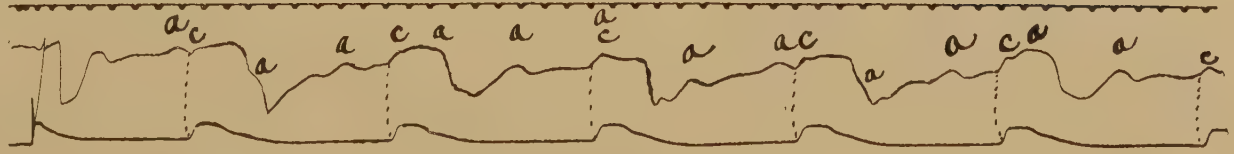


FIG. 219.

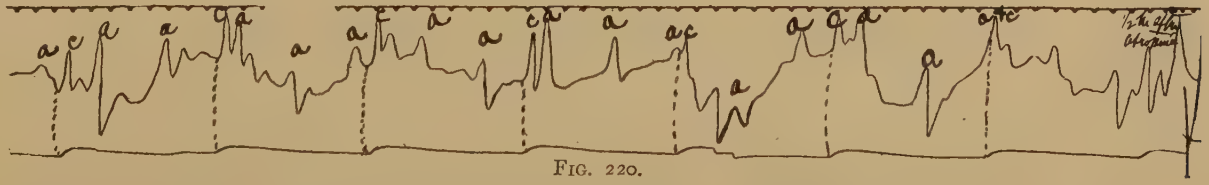


FIG. 220.

FIGS. 219, 220.—Complete heart block showing occasional superposition of *a* and *c* waves.

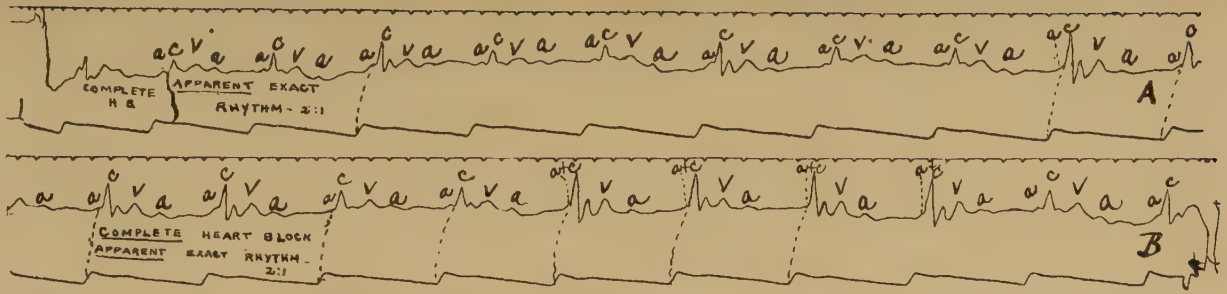



FIG. 221.—A.B., Complete block. In A the block is only apparently incomplete because the auricular speed is exactly twice that of the ventricle. In B, continuous with A, there is a slight variation in auricular and ventricular rates; the *a-c* interval becomes progressively smaller until *a* and *c* are superimposed, showing that the exact doubling of speeds and the apparently normal *a-c* interval in A were purely accidental.

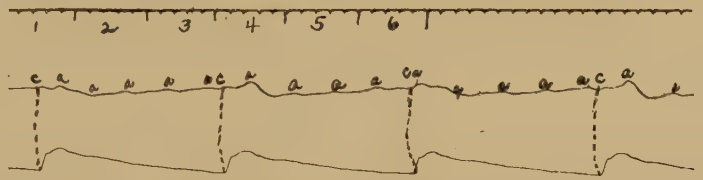



FIG. 222.—Complete block; ventricular rate 25 per minute; auricular rate 105 per minute. This tracing was corroborated by an electrocardiogram.

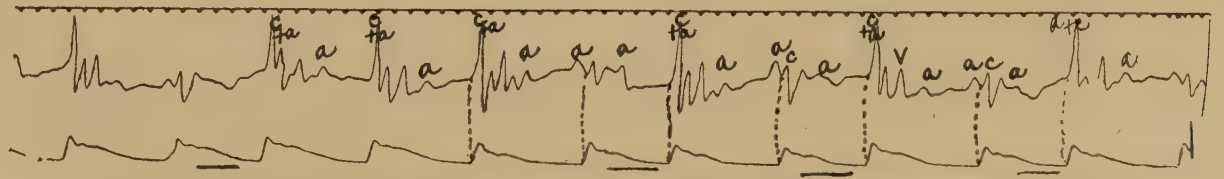


FIG. 223.—Complete heart block with irregular ventricular action. The shorter radial beats are underlined.

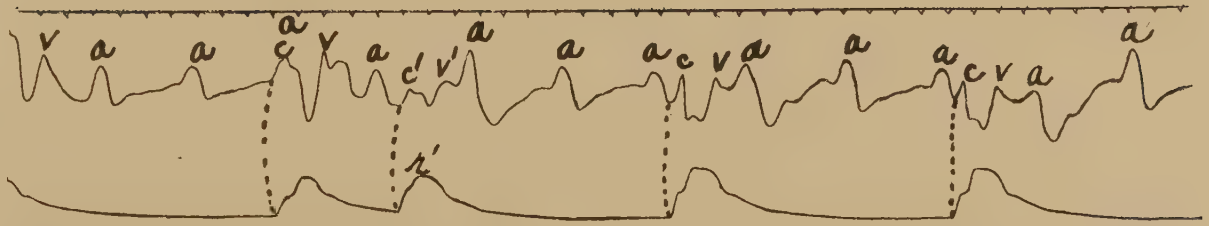


FIG. 224.—Complete heart block with a ventricular extrasystole ($r'$). The ventricular rate is 27; the auricular rate is 81 per minute. The pause following the extrasystole is not compensatory; it equals a rhythmic beat.

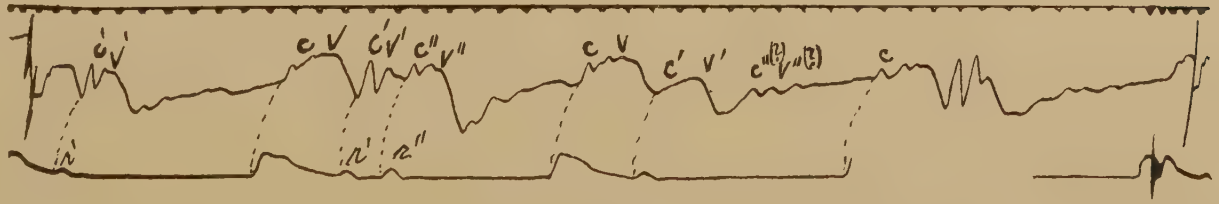


FIG. 225A.

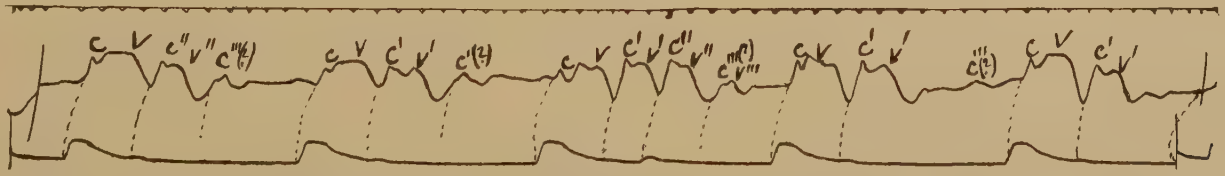


FIG. 225B.

FIG. 225.—A and B. Parts of continuous tracing. Heart block with auricular fibrillation. Some of the weaker heart beats are shown as small pulse beats at $r'$ and $r''$. Others are frustrane. Note the absence of $a$ waves in the phlebogram.

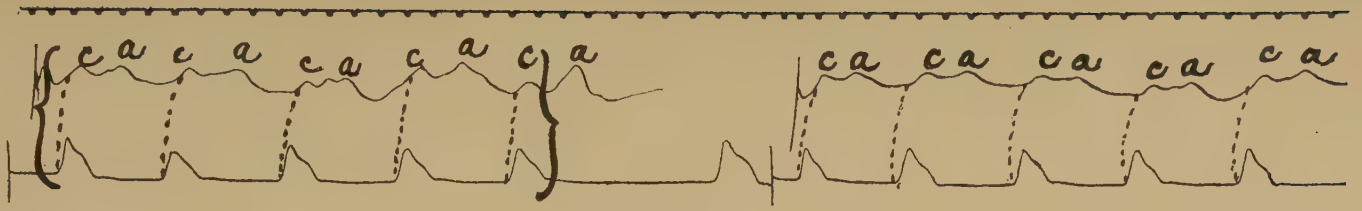


FIG. 226A. (1)

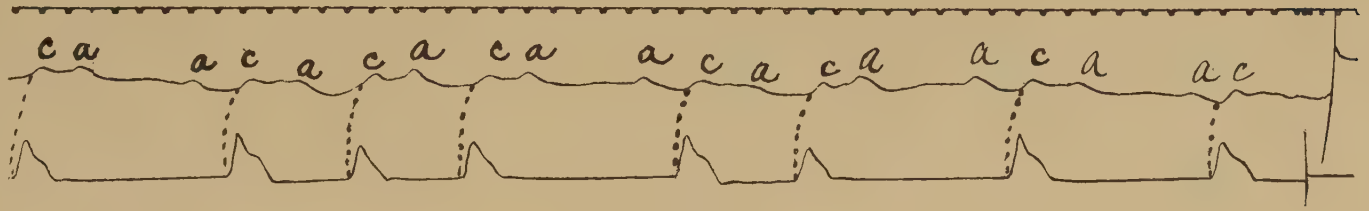


FIG. 226A. (2)

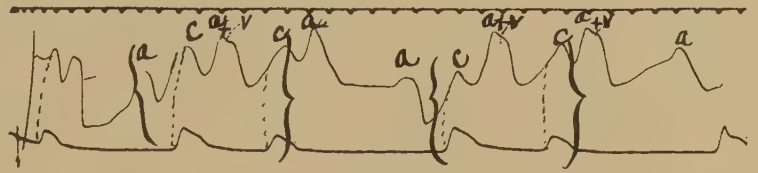


FIG. 226B.

FIG. 226.—A and B. Taken from the same patient on different days. (1) and (2) are continuous. There are two ventricular rates: 66 and 47 per minute. The auricular speed is constant at 66 per minute. The conduction time (*a-c* interval) of the faster beats (within brackets) is extremely prolonged (from 0.5 to 0.55 second). The slower beats apparently occur when the conduction time is so prolonged that the ventricle no longer answers the auricular impulse. The ventricle and auricle then beat at the same speed, the auricle following the ventricular contraction. Another possible interpretation is that the ventricle acts temporarily as pace-maker, and the auricle answers impulses generated in the atrio-ventricular node. (See Backward Conduction.)

per minute. Complete heart block represents the purest type of regular heart action. Polygraphically, the carotid (*c*) waves are identified in the usual manner. Since the auricular speed varies but slightly if at all, the auricular (*a*) waves are recognized by their occurrence at regularly spaced intervals (Figs. 217, 218). Because of their varying rates, the *a* and *c* waves occasionally fall simultaneously with, and are superimposed into, taller or broader peaks (Figs. 220–223). Figure 221 resembles incomplete block, because auricular and ventricular rates temporarily correspond. Examinations of longer sections, however, show that this ratio is accidental, for, with slightly varying auricular and ventricular speeds, there is at first closer approximation and finally superposition of the *a* and *c* waves (Fig. 221 B).

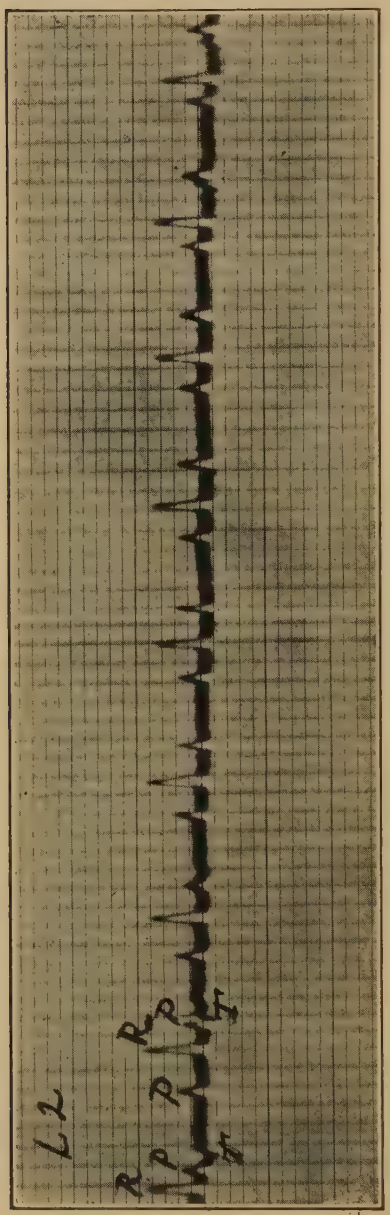



Fig. 227.—Complete heart block. The ventricular rate is 43, the auricular, 75 per minute.

In complete heart block, auricular activity occasionally is abnormally rapid, so that its ratio to ventricular beats is as four or five to one. While regular rhythmic activity is the rule in complete heart block, there are exceptions; as, for instance, slight variations in the ventricular rhythm (Fig. 223). The regularity may also be disturbed by ventricular extrasystoles. These are usually single (Fig. 224, *r'*). The pause following them is not compensatory, but is equal in length to a rhythmic beat. Very rarely such extrasystoles are multiple (Figs. 225, A and B, *r' r''*).

The case illustrated by Fig. 225 was originally one of complete heart block with a rhythmic ventricular rate of 35, and an auricular speed of 72 per minute, as demonstrated by polygraphic and electrocardiographic tracings. The auricular beats could be plainly heard in the interventricular

FIG. 228.—A and B are parts of the same electrocardiogram. A shows incomplete block with a 2:1 rhythm. B shows complete block.

pauses. Through inadvertence on the part of the nurse, digitalis was administered for several weeks after having been ordered discontinued. The pulse then became arrhythmic. The auricular beats were no longer heard. "Extrasystoles" (*i.e.*, ectopic aberrant beats), most of which were frustrane, were heard at the apex. The tracing showed some of them registered in both the radial and jugular curves (Fig. 225, A, B, $r'$, $c'$, $a''$, $c''$); others, in the jugular only (Fig. 225, A, B, $c''$); regularly recurring auricular waves could not be identified. Digitalis was discontinued. After two weeks, electro-

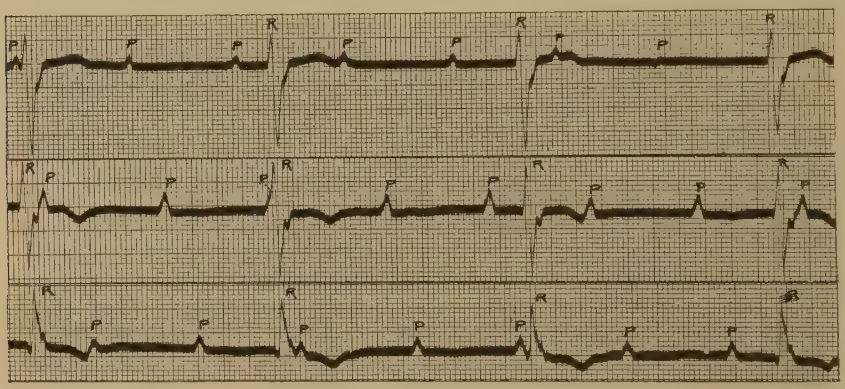


FIG. 229.—Complete heart block. The ventricular rate is 30; the auricular, 75 per minute. (*Courtesy of A. E. Cohn.*)

cardiographic tracings showed that the original type of heart block was present, and the auricular beats were again heard. From the ausculatory evidence, from the absence of auricular waves in the jugular, and from the fact that digitalis poisoning sometimes induces auricular fibrillation and "extrasystoles," it seems probable that the polygraphic tracing represents heart block, auricular fibrillation, and aberrant beats—a unique instance of digitalis poisoning in a patient with complete heart block.

Another rare example of peculiar disturbance of ventricular rhythm is that represented in Fig. 226 A and B, taken from a patient on successive days. Digitalis had not been given. The radial tracing showed ventricular arrhythmia due to abrupt and reciprocal changes from faster to slower rates. The faster rate was 66; the slower, 47 per minute. The auricular speed remained constant at 55. A study of the polygraphic tracing corresponding to the faster beats (included in the brackets) showed an extremely long conduction time, the *a-c* interval varying from 0.50 to 0.55 of a second. Occasionally the ventricle did not respond but contracted at the idioventricular rate (complete heart block). This condition existed for one week. Thereafter electrocardiograms taken frequently for a period of one year always showed complete block (Fig. 209), the auricular speed being 100 to 110, the ventricular 45 to 50 per minute. The block was uninfluenced by atropin injections.

Comparatively rapid ventricular rates are occasionally encountered in heart block. For example, in one of my cases, the ventricular speed was at one time as high as 77 per minute and the rhythm slightly irregular. The

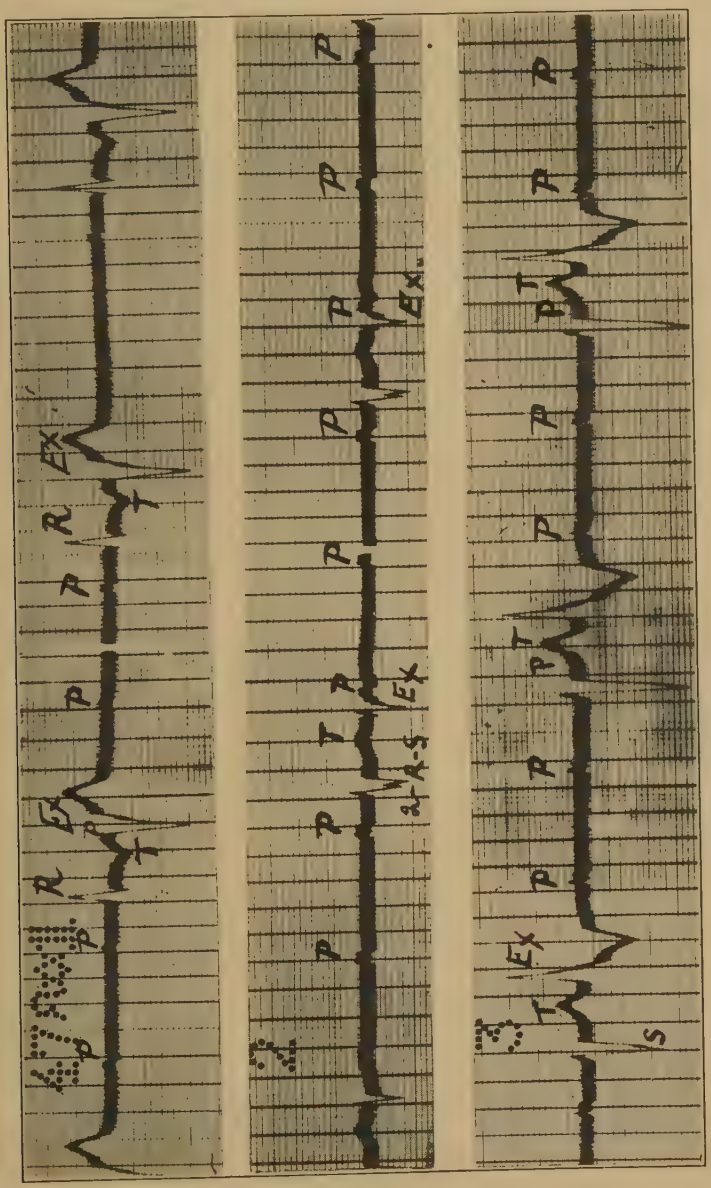


Fig. 230.—Complete heart block with extrasystoles (Ex.). The latter occur after every ventricular beat: The auricular rhythm (P waves) remains undisturbed.

following curves are examples of heart block with varying auricular and ventricular rates. Figure 227 is that of a man of 60 with myocarditis and aortitis, and shows complete heart block. Figure 228 is given in two parts:

A, illustrates incomplete block with a 2 : 1 rhythm; B, taken a few moments later, shows complete block. In section A the block only appears incomplete because of slightly varying auricular and ventricular speeds. This tracing was taken from a young man who had peritoneal tuberculosis with irregular fever and who, except for the arrhythmia, presented no sign of cardiac disease. The lungs were normal. The patient had been in the habit of feeling his pulse before his illness and had always found it of normal rapidity, that is, about 70 per minute. The peritoneal tuberculosis became quiescent following laparotomy. The heart block continued. Atropin was injected several times without influencing the block. Its only effect was a slight transient increase of the ventricular rate. The patient died two years later as the result of an accident. A necropsy was performed. In the abdomen, omental tuberculosis was found. The lungs were normal; there were no enlarged glands at the pulmonary hilus. On careful macroscopic examination, the cardiac valves and musculature were found normal. Although the atrio-ventricular conduction system has not been microscopically examined, there was no evidence of any gross lesion at the site of the bundle. In other words, there was no apparent pathological change in the heart itself to account for the block. This case will be etiologically discussed in another connection (Chapter XI).

Another case of complete heartblock is shown in Fig. 229. A case of heartblock with extrasystoles is shown in Fig. 230.

**Clinical Recognition of Heart Block.**—The ventricular rate in complete heart block is usually between 25 and 40, although rates as low as 8 per minute have been reported. The auricular beats are often heard in the apical region or at the third left interspace as soft, faint, distant sounds interspaced between the ventricular contractions. Synchronous with the sounds of the auricular contractions, jugular pulsations unaccompanied by carotid beats may be seen in the neck. There are no compensatory pauses in the regular ventricular action of heart block, thus distinguishing this arrhythmia from extrasystoles. Incomplete heart block with changing ventricular ratios, for example from 2 : 1 to 3 : 1, requires differentiation from sino-auricular block; this distinction is based chiefly upon the auricular contractions which are present and sometimes audible in the first type of arrhythmia, and upon the absence of auricular beats, visible or audible, in sino-auricular block It is sometimes possible to diagnose complete heart block merely from observation of the pulsation of the neck. Prominent pulsations indicative of the simultaneous contraction of auricle and ventricle (*a-c* wave), or of the auricular and the ventricular filling wave (*a-v*) can then be observed. Similar large jugular waves, sometimes seen with extrasystoles (*c'v'*, *a'c'* or *c'a* waves), are differentiated from those in heart block by the slow ventricular rate of the latter.

**Pulse Alteration—Pulsus Alternans.**—This is a disturbance, not of rhythm but of strength of the ventricular contractions, and therefore of the

FIG. 231.—Normal rhythm; slight alternation.

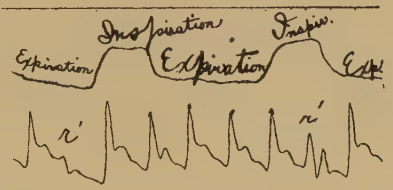
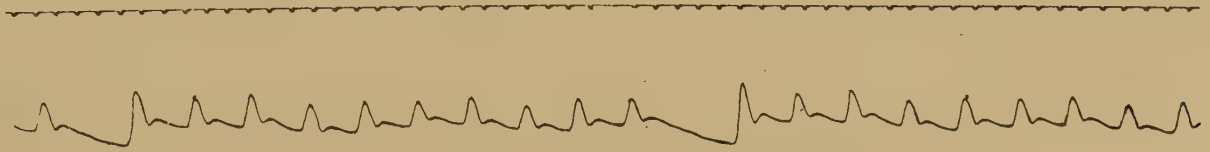



FIG. 232.



FIGS. 232, 233.—Alternation following extrasystoles.

pulse beats; strong beats regularly alternate with weaker ones. Alternation of the pulse is often not sufficiently marked to be detected by palpation (Figs. 231, 232), hence the importance of radial tracings for its diagnosis. It may be occasionally diagnosed by observing alternating difference in the systolic blood pressure readings when making blood pressure observations. It occurs in tachycardia, especially when paroxysmal, and results then directly from the ventricular acceleration. Alternation is also fairly common after extrasystoles (Figs. 232, 233).

Alternation is usually found in patients with dangerously weakened myocardium and is commonly regarded as of grave prognostic import. Indeed, most patients with heart disease and decompensation who have an alternating pulse die within one year. However, alternation is occasionally found in patients with normal hearts, or as a result of digitalis medication. The mechanism of alternation is still the subject of many conflicting opinions and theories. The condition has been ascribed to disturbance of the function of contractility; to the fact that not all the ventricular fibers contract with the smaller beat; or because the systoles of the stronger are of longer duration than those of the weaker beats, thus encroaching upon the rest period of the latter. Electrocardiographic tracings of patients with alternation have partly upset some of these hypothesis. For example, there is no evidence that varying amounts of ventricular musculature are involved or that the path followed by the weaker differs from that of the stronger contractions; nor is there any difference in duration of their contraction times. Einthoven suggested an explanation similar to that involved in the irregular pulse excursions of auricular fibrillation, namely, that the increased blood pressure of the stronger contractions acting upon the weakened myocardium prevents the heart from emptying itself at the next systole, and consequently produces a smaller pulse. This theory puts the explanation a step nearer, but scarcely explains the *rhythmic* alternating regularity in pulse pressure.

**Pulsus Paradoxus.**—This irregularity is indicated by a gradual waning and waxing in the strength of the pulse beat with respiration, but without any change of rhythm. It has been regarded as characteristic of pericarditis with effusion. I have found it especially frequent in cases of severe myocardial insufficiency. Occasionally the pulse becomes so weak and small as to be scarcely perceptible on palpation.

**Hemisystole** is mentioned for the sake of completion of the subject of arrhythmias. It refers to inaction of one ventricle at the same time that the other is in systole. One can scarcely conceive how, in any pumping mechanism like the heart with its well-balanced pulmonary and systemic circulation, the circulation can be carried on by one ventricle without its fellow for any appreciable length of time. Up to the present, there has not been reported any undoubted clinical case of hemisystole.

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## CHAPTER XI

## THE ARRHYTHMIAS—THEIR CLINICAL ETIOLOGY AND THERAPY

At the outset it is important to emphasize that, although certain types of arrhythmias may be associated with various pathological entities, almost any irregularity may be the result of non-organic, neurogenic, so-called "functional" causes. From the standpoint of etiology, therefore, the primary essential is to discover, if possible, whether the arrhythmia is of functional or of organic origin. Therapy will be only briefly indicated here; full details will be found in the chapter on Circulatory Remedies (Chapter XX).

**Sinus Arrhythmia.**—As a physiological phenomenon, sinus arrhythmia is quite common not only in children and young adults but also in the middle-aged. It is, however, also present in organic heart disease. I have found it for example in young individuals suffering from aortic and mitral disease. The hearts of these patients were compensated; they had not received digitalis. Possibly this arrhythmia in patients with heart disease can be regarded as a favorable sign, since it is an evidence of physiological vagal control. Sinus arrhythmia is also frequent as a phenomenon after febrile respiratory catarrah terminating by critical defervescence. The arrhythmia is then regarded by many observers as the result of toxic or infectious myocarditis. Its innocent nature, however, is proven by its disappearance within a few days, and by the absence of all signs of cardiac disease. Sinus slowing is sometimes a purely pathological occurrence; it then bears no relation to breathing. For example, it occurs in severe secondary anemia following sharp hemorrhage. I have observed two such cases. In both, the sensorium was clear and the arrhythmia gradually disappeared with improvement in the anemia. In such instances, I believe sinus arrhythmia is due to nutritional changes in the sino-auricular node, or to increased vagus inhibition on the part of the cardio-inhibitory center. In another case, an adult whose heart at necropsy showed marked cardiosclerosis involving the endocardium, mitral valves, aorta and the coronaries—the pulse ranged between 50 and 60 per minute during the last year of life. Polygraphic tracings showed sinus-arrhythmia. Microscopic examination of the sino-auricular node was not made; sclerotic changes in that region may have been the etiological factor.

Pathological sinus arrhythmia, or sinus arrhythmia encountered in febrile crisis, requires no medication. When due to the other serious etiological factors above mentioned, treatment should be directed to them rather than to the arrhythmia itself.

**Sino-auricular Block.**—This is usually the result of digitalis poisoning. It has also been produced by pressure over the vagus in the neck, by pressure upon the eyeballs (oculo-cardiac reflex) in human beings, and by various

experimental procedures in animals. It is occasionally the result of tobacco poisoning (Figs. 208, 209). Otherwise it is exceedingly rare as a clinical phenomenon. The condition itself requires no medication unless accompanied by dizziness; atropin sulphate is then sometimes of value. At the beginning the dose should be small, gr. $\frac{1}{250}$ three times daily before meals. The amount should then be increased up to the individual's physiological tolerance.

**Extrasystoles.**—Although there are many exceptions, auricular extrasystoles are usually of functional origin, while the ventricular are often of organic nature. When accompanying organic disease, auricular or ventricular extrasystoles may mark the beginning of decompensation and then remain as permanent arrhythmias. In acute heart failure, they are sometimes the forerunners of a fatal termination. The onset of exacerbations of acute endo- or pericarditis may be accompanied by extrasystoles. We do not know the exact manner in which organic cardiac lesions increase ventricular excitability and thus produce premature contractions; it seems probable, however, that impaired cardiac nutrition plays an important role when severe cardiac disease is present.

When extrasystoles are found in cardiac failure, treatment of the latter (Chapter XIV) is the prime consideration. With the establishment of cardiac compensation, the extrasystoles usually disappear; when caused by endo- or pericarditis, the therapy consists mainly of salicylates, preferably sodium salicylate in large doses. My usual procedure is to give 10 grs. hourly for six doses unless tinnitus occurs. Thereafter, the same dose is given three times daily. Occasionally the addition of bromides is of value.

**Functional Extrasystoles of Extracardiac Origin.**—Cerebellar and cerebral conditions which exert pressure directly or indirectly upon the cardio-inhibitory center can cause premature contractions. Such pressure factors apparently act by direct excitation of the vagus center. Thus a patient with cerebellar tumor and normal circulatory apparatus had occasional extrasystoles during the last few months of life. That they were due to pressure was demonstrated at operation for the removal of the tumor. The latter was deeply seated, each attempt at its removal being accompanied by premature contractions. Acute gastric diseases with vago-excitive characteristics (belching, pyloro- and cardiospasm), less often intestinal disease, is occasionally accompanied by extrasystoles, usually auricular in type. Their etiology has been variously explained. The usual theory is that, after a meal, the distended stomach presses the diaphragm against the heart, thus embarrassing circulation. Another reason given is the action of absorbed toxic products upon the neurogenic cardiac control. In the majority of cases that came under my observation, the extrasystoles occurred most frequently when the stomach was empty. Several of these patients, fluoroscoped after a barium meal, showed no gastric distention or other evidence of abnormal pressure against the diaphragm; in fact, their stomachs

were contracted, and hypermotility was present. These observations nullify, I believe, the commonly advanced theories. The intimate physiological relationship between the nerves of the stomach and those of the heart lead me to believe that extrasystoles accompanying gastric disturbances are probably caused by reflex excitation of the cardiac nerves but the path followed by the reflex arc is not clear.

Acute inflammation of the gall-bladder and bile ducts, with or without gastric symptoms, may also be accompanied by premature contractions. Although the theory is based upon insufficient data, toxemia has been assumed as the etiological factor for such extrasystoles. The arrhythmia is often present very early in the disease, when extensive toxic absorption appears improbable. Similar to the views expressed with reference to extrasystoles in gastric disturbances, I believe that most cases occurring in gall-bladder disease are caused by reflex excitation of the neurogenic control of the heart, although at present the centripetal nerve-path involved is unknown.

Peritonitis is another disease in which premature contractions may occur. One case of appendicitis, followed by general peritonitis and death in an elderly individual with cardiosclerosis, had ventricular extrasystoles at the onset of the attack; during the last few days of life, auricular fibrillation was present. It was impossible to decipher the varying roles played by toxemia and reflex nerve excitation in the production of these arrhythmias; in this case I believe both were factors.

The onset and crisis of acute catarrhal febrile affections are frequently marked by premature contractions. The latter are generally regarded as evidence of toxic myocarditis. This relationship seems doubtful for several reasons. Most of the cases I have observed were not toxic: For example, the extrasystoles occurred with mild grippe and tonsillar affections in patients who scarcely felt ill. The arrhythmia was found at the crisis or immediately thereafter, that is, when the severe symptoms had disappeared. There were no circulatory symptoms. On clinical grounds, therefore, it seems probable that such extrasystoles are due to abnormal products (toxins?) flowing in the general circulation and affecting the cardio-inhibitory center, but not the myocardium.

Regarding extrasystoles in pneumonia, it should at the outset be stated that this disease can insidiously cause severe cardiosclerosis (Chapter V); but this does not apply to premature contractions occurring in the acute stage of pneumonia. Unless circulatory symptoms are already present, pneumonic patients with critical or post-critical extrasystoles do not suffer from cardiac failure as a result of the arrhythmia. The latter usually disappears within a few days without any treatment. If myocarditis or other severe pathological damage to the heart caused these extrasystoles, symptoms of circulatory failure, instead of being absent, would be prominent. The present theory regarding crisis in pneumonia is that the system is at such times suddenly flooded with toxins elaborated in the pneumonia area. That the

cerebral centers become involved is shown by critical sweats and vasomotor symptoms. Correlating these observations, it seems most likely that such extrasystoles are the result of pneumonic toxins acting on the cardio-inhibitory center.

Among drugs, the digitalis bodies, less frequently the salicylates, produce extrasystoles. When the former are given the arrhythmia is usually coincident with the full therapeutic effect of the digitalis. Premature contractions are common in hypertensive diseases (Chapter XVII). Fright, nervousness and epileptic seizures, overindulgence in coffee, tea and tobacco, are additional causes of premature contractions. Extrasystoles also occur in conjunction with the vaso-motor symptoms of the climacterium.

There still remains a group of patients in whom no cause for extrasystoles can be discovered. It is interesting in this connection to note that functional extrasystoles, even though they later disappear, are apt to recur after any slight disturbance (for example, overexertion, acute indigestion) which reflexly affects the normal inhibitory control.

Finally, it should be emphasized that extrasystoles can occur in perfectly healthy individuals, young and old, in whom there is no evidence of cardio-vascular disease. Frequently these individuals are not cognisant of any pulse irregularity. The fact that individuals with healthy hearts can have premature contractions had long been disputed, but its occurrence in them is by no means infrequent, and examination of such cases years later shows no evidence of cardio-vascular disease.

**General Therapy for Extrasystoles.**—Such underlying diseases as gastric disturbances, appendicitis, etc., naturally require their appropriate remedies, and the presence of extrasystoles in no wise affects the usual therapeutic indication and procedures. Unless extrasystoles of reflex or neurogenic origin cause such subjective sensations as "fluttering in the chest," transient faintness, etc., or are in themselves causes of circulatory failure (an exceedingly rare occurrence), they require no therapy. When medication is indicated, the bromides are of value, the dose is 15 to 30 grains, preferably given once daily. Another excellent drug for quieting the nervous system is luminal. The usual dose is 1½ grains given once every morning. Atropine sulphate in small doses (gr. $\frac{1}{200}$ to $\frac{1}{100}$ three times daily) is of use when the extrasystoles are due to reflex gastro-intestinal disturbances. I have found the solid extract of suprarenal gland given in tablet form in one or two grain doses of special use when the extrasystoles are accompanied by vaso-motor disturbances. Digitalis has occasionally been advised because of its effect in increasing vagus inhibition.

**True Bradycardia.**—The term has already been defined (Chapter X). It may appear as a rare congenital anomaly in patients with normal hearts and circulation. A sequential rhythm and pulse rate between 45 and 60 per minute is sometimes encountered in senile cardiosclerosis. Most cases of true bradycardia are of extracardiac origin. Patients with lead colic, and

gastrointestinal disorders accompanied by abdominal pain, belching or diarrhoea, are apt to have a slow pulse rate. This is probably due to reflex vagus inhibition from excitation of gastric and intestinal nerves.

Catarrhal jaundice is not infrequently accompanied by true bradycardia, and is then usually ascribed to toxic myocarditis. In the patients whom I have observed, there was no evidence of the latter disease. The bradycardia in these cases may be ascribed to two causes: In those with cholemic symptoms it is probably of central inhibitory origin while in non-toxic cases with painful local symptoms, it is due, I believe, to reflex vagus inhibition from abnormal excitation of the nerve filaments surrounding the gall bladder.

**Appendicitis.**—Slight bradycardia with pulse rates between 60 and 65 per minute is occasionally encountered in this disease. Myocardial involvement or quiescence of the appendical lesion has usually been assumed as the cause. Unless general peritonitis and severe toxemia are present the first factor—myocarditis—seems improbable. Regarding the second, it appears more likely that a slow pulse with temperature is an indication of continued and active excitation at the inflammatory focus rather than an evidence of the latter's quiescence.

Among drugs, the digitalis bodies and salicylates can cause a slow pulse by increasing vagus inhibition.

Epileptics occasionally have an abnormally slow pulse. This is also true of those types with so-called vago-vasal attacks (Gowers) in which there are no convulsions, but vaso-motor symptoms, vomiting, syncope and pains along the distribution of the intercostal nerves are present.

A slow pulse is also fairly common at the crisis of pulmonary affections, or during the course of the disease. When present at the crisis, I believe it is due to the neurotropic effect of toxins liberated during critical defervescence.

Therapy for bradycardia is only indicated when the slow pulse rate is accompanied by subjective symptoms. Atropine is then of most value, given first in small, later in increasing doses, until the full physiological effect has been obtained.

CLINICAL AND EXPERIMENTAL CAUSES OF AURICULO-VENTRICULAR HEART BLOCK

I. Cardiac causes.
- (*a*) Destruction of the A-V bundle or auriculo-nodal junction by tumors (benign or malignant), calcareous deposits, fatty and inflammatory infiltration, fatty degeneration, coronary infarct, hemorrhage in the node, fibrosis.
- (*b*) Severe myocardial degeneration. Destruction of the branches or terminal arborizations of the conduction system.

II. Extra-cardiac factors.
- II. Drugs (digitalis—morphine—nicotine(?).
- III. (*a*) Asphyxia.
- (*b*) Chemical poisons { Producing acidosis, toxins, anaphylatoxins.
- IV. Interference with cerebral circulation.
- V. Abnormal pressure on the cardio-inhibitory center.
- VI. Abnormal pressure on the vagus.
- VII. Increased vagal inhibition.
  - (*a*) Digital pressure on the vagus.
  - (*b*) Peripheral excitation.
- VIII. Endocrine disturbances.

Heart block is here used to denote auriculo-ventricular dissociation, complete or incomplete; it does not refer to lengthened conduction time, blocked auricular beats, or to sino-auricular block. In order to establish a certain coherence between the diverse etiological factors concerned in heart block, I have made and adopted the etiological classification given above, which probably includes the great majority of known factors. Experimental causes are also mentioned because they may furnish an insight into some clinical cases which have experimental analogies.

I. (*a*) Any lesion which completely severs or destroys the main branch of the conduction system produces complete heart block. If the destruction is incomplete, there may be enough healthy strands left to carry impulses from auricle to ventricle. A suddenly deficient coronary supply, the result of thrombosis, infarct or disease of the nutrient artery of the bundle of His, can so profoundly disturb the nutrition of the conduction system as to produce transient or permanent heart block.

(*b*) Just as fibrotic and calcareous degeneration in the conduction system is almost invariably accompanied by gross pathological changes in the ventricular musculature, so in extensive cardiosclerosis there are often fibrotic and other degenerative changes in the connecting bundle. Even if the bundle remains sufficiently normal to convey auricular impulses to the ventricle, cardiosclerosis in itself can probably produce heart block by destruction of the branches or terminal arborizations of the conduction system in the ventricular musculature (Chapter I) so that impulses passing through the bundle are prevented from reaching their final destination in the ventricles. And even if these final conduction strands (terminal aborizations) are sufficiently normal to carry impulses to their final ventricular distribution, one may still conceive a pathological condition in which the ventricular musculature may be so diseased as not to be capable of normal response. Any or all of these causes (Group I (*b*)) may operate in those cases of heart block in which the main conduction system is found quite or fairly normal upon pathological examination. The causes would then lie either in the subsidiary branches of the conduction system or in widespread myocarditis.

**Therapy.**—When syphilis is the etiological factor, vigorous antiluetic treatment—salvarsan, bichloride injections, and iodide of potash is indicated (Chapter XVI). Before intravenous medication with neosalvarsan, arsphenamin or salvarsan is begun, it is advisable to start with a few injections of mercury. One should also start cautiously with intravenous medication, beginning with about one quarter of a full dose and then gradually increasing every week until the full dose is reached. The advisability of using digitalis and atropin in cardiac disease with heart block is discussed subsequently. A few cases of heart block have been reported in which the administration of thyroid extract was followed by beneficial results.

II. Digitalis can produce complete or incomplete block. This arrhythmia has not only been observed in those with heart disease who had received

digitalis but it has also been induced by digitalizing healthy hearts. Complete heart block has also been induced by the injection of morphine in dogs, and was regarded as an effect upon the cardio-inhibitory center. Morphine poisoning in man is sometimes accompanied by very slow pulse rates. These have never been graphically recorded; they may be due to the same cause found in the animal experiments; namely, heart block. Tobacco poisoning is also occasionally accompanied by a very slow pulse. To determine its precise nature, graphic tracings are necessary. In this manner it may in the future be demonstrated that some of these tobacco arrhythmias are likewise due to heart block.

III. (*a*) In animals, the experimental production of asphyxia is accompanied by varying degrees of heart block; the essential cause is assumed to be nutritional disturbance from lack of oxygen in the junctional tissue. With our present imperfect knowledge of the chemistry of the blood, it is impossible to state whether other chemical factors (Group III (*b*)) can act similarly in the production of heart block by profoundly interfering with cardiac nutrition. Such factors are abnormal constituents in the blood causing lessened blood alkalinity (acidosis); chemical poisons elaborated in infectious diseases (toxins); and anaphylotoxins. Regarding acidosis, I have several times observed a peculiar form of heart block in the terminal stages of patients suffering from cardiosclerosis and nephritis, in whom electrocardiograms showed very rapid and irregular auricular and ventricular activity (Terminal Arrhythmias, Chapter X). Cyanosis was not a constant factor. Blood examinations for non-protein nitrogen and other products were not made, but from the clinical syndrome, it seemed that retained chemical poisons constituted the essential cause of the arrhythmia. With reference to toxins, it has been shown experimentally that transfused pneumonic blood profoundly impairs cardiac contractibility. I have had occasion to study two cases which have a bearing upon these experimental observations. Both developed heart block during the course of pneumonia. In one an autopsy was performed. Upon macroscopic examination the heart was found normal. Careful microscopical examination of the conduction system also showed that it was normal. Such clinical and experimental observations indicate that in pneumonia, at any rate, toxins have an action not only upon the cerebral centers, but also locally upon the heart. Block may then result from interference with nutrition of the A-V conduction system alone or in conjunction with the remainder of the cardiac musculature.

Complete heart block has been produced in animals by experimental anaphylaxis; the cause is assumed to be due to an effect upon the heart itself.

**IV. Interference with Cerebral Circulation.**—This factor probably operates by interference with nutrition of the cardio-inhibitory center. Sclerotic change in the arteries at the base of the brain is the pathological condition most frequently observed. In one reported case of heart block, few changes were found in the conduction system or cardiac musculature,

the cerebral arteries constituting the circle of Willis were markedly diseased. The brain itself was normal. Tumors and other pathological conditions which impede cerebral circulation may have a similar effect in producing heart block.

**V, VI. Abnormal Pressure on the Cardio-inhibitory Center and Vagus.** Tumors and adhesions affecting the vagus or even the cardio-inhibitory center are the most likely pathological entities which can produce heart block. One case of heart block from pressure on the vagus has been reported; a large tumor involving the nerve in the anterior mediastinum was found.

**VII. Increased Vagal Inhibition.**—It has been shown that in children with heart disease, digital pressure on the vagus in the carotid sheath may be followed by temporary heart block. This manoeuver probably acts by causing increased vagal inhibition. A case has also been reported in which swallowing induced heart block in a patient with delayed conduction time. These are examples of induction of this arrhythmia from direct excitation of the vagus in susceptible individuals.

(*b*) **Reflex Peripheral Excitation of the Vagus.**—I have observed cardiac inhibition with the production of complete heart block, apparently reflexly evoked by peripheral stimulation of the pneumogastric branches supplying the stomach. The clinical history of the patient showed that the pulse had been normal before the onset of tuberculous peritonitis from which the patient suffered when he first came under observation. For several weeks high temperature and abdominal pain had been present. The abdomen was opened, and fluid evacuated. Some weeks after operation, the symptoms of tuberculous peritonitis entirely subsided. Two years later the patient was killed in an accident. Complete heart block, as shown by numerous polygraphic and electrocardiographic curves, was present before and after the operation, and up to the time of the patient's death. The presence of the arrhythmia caused no symptoms. A necropsy was performed; the lungs were found normal; a mass of enlarged suppurating tuberculous glands adherent to stomach and intestines was found. Careful macroscopic examination showed the heart to be absolutely normal. Microscopic examination of the conduction system was not done but careful scrutiny showed the absence of any gross lesion. Toxemia as the cause of the heart block could be excluded, for the patient was clinically well, had had no temperature, and was at work for months prior to his death. Weighing the pathological and clinical data it seems probable to me that the heart block was due to abnormally increased vagal inhibition reflexly excited by involvement of peripheral nerve filaments in the abdominal tuberculosis.

**VIII. Endocrine Disturbances.**—I have seen complete heart block in an individual who presented the typical syndrome of a mixed endocrine disturbance (so-called pluriglandular syndrome); there was no evidence of a cerebral new growth. Aside from the arrhythmia, the cardio-vascular examination presented nothing abnormal. Our knowledge of endocrinology

is at present too limited to discuss the possible etiological factors concerned in heart block of this type.

**General Therapeusis of Heart Block.**—Therapeutically, atropine sulphate should be tried in all cases of heart block; it should be given hypodermatically until the full physiological effect is reached. As much as $\frac{1}{50}$ or even $\frac{1}{25}$ of a grain may be given in the effort to remove the block. Should block be thus removed, atropine is to be continued three times daily in doses from gr. $\frac{1}{150}$ to $\frac{1}{100}$. Recently the extract of thyroid gland (the alpha iodin fraction of Kendall) has been advised in order to increase the ventricular rate in heart block. The drug is to be pushed until the full physiological effect is reached. I have had no personal experience with the drug; if given cautiously and begun with small doses, I see no harm in its trial. When possible, appropriate treatment should be directed toward the individual etiological factors and toward the clinical symptoms. This applies particularly to the usual decompensation so frequently present in heart block, and should be treated in the usual fashion with digitalis, caffein, etc. (Chapter XX). In some cases of incomplete heart block, however, special caution in the use of digitalis should be observed (Chapter XI).

**Prolonged Conduction Time.**—Among drugs, digitalis occasionally produces increased conduction time from auricle to ventricle. The chief cardiac cause of this arrhythmia is myocardial degeneration; indeed, a prolonged *a-c* interval in the polygram or a prolonged P-R interval in the electrocardiogram (Chapter X) may be the only evidence of this disease. Prolonged conduction time is encountered fairly frequently during the course of, or immediately after, attacks of rheumatic endocarditis. It is then assumed to be due to myocardial involvement. It is also met with in acute or sub-acute nephritis in which there are none of the usual clinical manifestations of myocarditis. In addition, prolonged conduction time is occasionally of neurogenic origin. Therapy is indicated for the symptoms of myocarditis or for other etiological factors, but not for the prolonged conduction time: The latter itself causes no symptoms and requires no medication.

**Shortened Conduction Time.**—I have found a shortened conduction period from auricle to ventricle especially frequent in exophthalmic goiter (Chapter X). The fact that experimental excitation of the right accelerator nerve is accompanied by a slightly, and excitation of the left accelerator by a considerably, shortened conduction time (Chapter I) is doubtless of etiological significance in exophthalmic goiter, in which the chief symptoms are due to hyperexcitation of the sympathetic system. Tachycardia is usually present with a shortened auriculo-ventricular conduction time. Tachycardia with its other clinical manifestations may then require medication, the shortened conduction time as such does not.

**Auricular Fibrillation.**—This is the usual arrhythmia accompanying decompensation in rheumatic mitral stenosis in the young and middle-aged. It is rare in valvular disease affecting the aorta. It is especially frequent in

the advanced cardio-sclerotic changes present in the old. It is also sometimes found in children; I have seen isolated instances in patients between 10 and 12 years of age with advanced valvular disease.

While auricular fibrillation is often a permanent arrhythmia; (indeed, it was formerly regarded as always permanent, hence the old term, perpetually irregular pulse) it is occasionally transient or it may even occur only in attacks. (See Cardio-vascular Clinics.) I have observed such attacks lasting several days in two cases of aortic aneurism. In one, it occurred with the gradual onset of severe decompensation; in the other, there was no discoverable cause for the attack. Another instance of transient auricular fibrillation was that of a woman of 60, a sufferer from mild myocardial insufficiency, who had been operated upon for empyemia of the gall bladder. Following the operation, there were several distinct attacks of broncho-pneumonia; the onset of each was marked by a moderate rise of temperature and by auricular fibrillation lasting one day. In mitral stenosis, *pari-passu* with fresh exacerbations of endocarditis, auricular fibrillation may occur. Thus, in a man of 45 with a double mitral lesion, from whose blood a non-hemolytic streptococcus was isolated, each sharp febrile invasion was accompanied by an attack of auricular fibrillation; these attacks lasted several hours or days. Patients with mitral disease who suffer from acute febrile disturbances of non-rheumatic origin are also prone to attacks of this arrhythmia; these may last throughout the fever. I have seen two such instances: One, a patient with mitral regurgitation, the other with mitral stenosis; both developed fibrillation during erysipelas. Auricular fibrillation may also be an initial symptom of coronary embolism or thrombosis (Chapter XXIII).

From these observations it is evident that in diseased hearts any additional insult to the endocardium, myocardium, or coronary arteries may be accompanied by auricular fibrillation. The permanence of the arrhythmia may depend upon the severity or permanence of the pathological damage.

The sovereign remedy for this irregularity when found in decompensated cardiac disease is digitalis. In some types, quinidin sulphate may also be employed. In many cases both digitalis and quinidin may be administered at the same time. The details of the administration of these drugs is elsewhere described (Chapter XX). I have occasionally added large doses of bromides or luminal (Chapter XX), or have initiated digitalis medication with one or two large doses of morphine when patients were very dyspnoeic or restless.

Auricular fibrillation also occurs, though very rarely, in individuals with normal hearts. For example, I have observed several cases of temporary auricular fibrillation coming on at the critical defervescence of lobar pneumonia; there was no evidence of heart disease or heart failure during the entire course of the disease or during convalescence. Some of these patients were examined months after the pneumonia; their hearts were found perfectly normal. Temporary or permanent auricular fibrillation is also fairly common in exophthalmic goiter. Apparently influences affecting neurogenic cardiac

control are essential factors. I have likewise seen auricular fibrillation in a heavy smoker whose heart was normal. A case due to hydrogen sulphid poisoning in a man with a normal heart has also been reported.

In those cases of fibrillation which are apparently of toxic or of neurogenic origin, I have found very little value in digitalis; the pulse or ventricular rate was not appreciably affected; dyspnoea, if present, continued. Such cases may perhaps be especially benefited by quinidine (Chapter XX). Our experience with this new drug has not yet been sufficiently large to state dogmatically in what type of case quinidin is apt to be of especial value. Large doses of bromide or luminal alone or combined with small doses of codeine seem occasionally of value.

**Simple Tachycardia—Ordinary Pulse Acceleration.**—Rapid, regular pulse rates usually range between 110 and 160 per minute, and are the result of excitation of the accelerator nerves. The causes are manifold. Excitement, fright, overexertion, fever are some of the commoner. In exophthalmic goiter, tachycardia is one of the cardinal symptoms. Gastro-intestinal disturbances of functional or organic nature are frequently accompanied by rapid heart action. Persistent tachycardia may be the only clinical evidence of a fresh exacerbation of an old endocarditis. Cardiac decompensation from any source is often accompanied by moderate tachycardia, and is sometimes the forerunner of other arrhythmias. Among other causes of rapid heart action are pulmonary tuberculosis, dyspnoea from pulmonary disease, tabagism, overindulgence in tea and coffee, and atropine.

Pulse acceleration requires medication only when the pulse rate is high or when subjective sensations are present. Even then, drugs are not of much value unless the primary disturbance in the stomach, lungs, etc., can be controlled. When tachycardia is due to fever, digitalis has no effect in reducing the pulse rate. The bromides or codeine are occasionally efficacious in slowing the heart in such cases. Digitalis may decrease the pulse rate when the tachycardia is due to cardiac decompensation.

**Paroxysmal Tachycardia.**—This cardiac irregularity is more often of functional than of organic origin. Its most frequent extracardiac cause is acute indigestion; the paroxysm is apparently brought on by excitation of the accelerators. For example, I have seen several patients in whom every attack of indigestion was accompanied by this arrhythmia. Another instance is a woman of 50 with abdominal cancer, gastric symptoms and severe secondary anaemia; with her, any slight fright or nervous excitement would frequently initiate an attack. At necropsy, the cardiac musculature was somewhat pale, otherwise the heart was normal. In another patient, a woman of 45 with a luetic history and positive blood Wassermann, sudden gastric attacks also initiated paroxysmal tachycardia. It is interesting to note that during these attacks there was marked dilatation of the left pupil, presumably an evidence of sympathetic nerve excitation. Other cases are described in Cardio-vascular Clinics.

Before medicinal treatment is begun with gastric sedatives, etc., the first therapeutic procedure should be firm pressure over the vagus in its course in the carotid sheath in the neck. Pressure on the right side should be first tried; if this does not succeed, pressure should be exerted over the left vagus. There are very few if any procedures in medicine more dramatic than the immediate cessation of an attack of paroxysmal tachycardia by vagus pressure. The antecedent discomfort, the sensation of fluttering in the chest, the dyspnoea, stop at once; the rhythm returns to normal, the attack ceases. The attack rarely recurs at once even if the underlying cause (fright, indigestion, etc.) be not entirely under control. There are a few important little points which must be remembered in the use of vagus pressure. The patient should be reassured and be told that the finger will be pressed into the neck for a moment or two; that the pressure will be somewhat painful but that it will not have any tendency to choke him or increase his shortness of breath. The patient is then asked to lay as flat and as comfortable as possible. The index finger is then placed lightly over the carotid at about its middle point in its course in the neck so that the carotid pulsations can be distinctly felt. Then, sudden, sharp pressure on the carotid (and vagus) is made directly backward, and the pressure continued for a minute or two. The finger pressure is then *slowly* relaxed. I believe that the effect of vagus pressure is somewhat enhanced if at the moment that vagus pressure is begun the patient is able to take a deep breath and will hold it as long as he comfortably can.

In one instance of paroxysmal tachycardia, I tried simultaneous pressure over both vagi in the neck when the paroxysm was not controlled by alternate right and left vagus pressure, respectively. The case is of sufficient interest to warrant a brief report of the history as well as the unique therapeutic procedure. M. T., age 45, female, unmarried, regular menses, has had attacks of paroxysmal tachycardia for many years. Between the attacks she has no cardiac symptoms of any kind. The paroxysm lasts from several minutes to several hours. She had scarlet fever when eight years old. The first attack is supposed to have started at about the age of ten. Since adolescence the paroxysms seem more frequent at the time of the menses. She is a rapid eater and has a tendency to bolt her food. Seasoned and heavy food causes gastric distension which is generally relieved by belching. Physical exertion itself has never initiated a paroxysm: For example, the previous summer she enjoyed ocean bathing without cardiac discomfort. The attack in which I first saw her was the worst she had ever had. It began in typical fashion. She suddenly felt a throb in her throat (very probably an extrasystole), immediately succeeded by tachycardia. The tachycardia was constantly present for three days with the ventricular rate regular and varying from 180 to 200 per minute. The bromides, chloral, morphine and digitalis were tried by her physician without any effect. Upon examination I found the systolic blood pressure 80, the diastolic, 70. Except for somewhat

distant heart sounds at the apex and rapid regular action, the heart showed no abnormality. There was passive congestion at the base of the left lung; the temperature was 101°, probably the result of the pulmonary stasis. The patient was dyspnoic and somewhat cyanotic; there was slight edema of the legs. I first tried sharp pressure over the right vagus; this was followed by a normal cardiac rate for several beats. The tachycardia then continued as before, and thereafter was not influenced by several other attempts at pressure over the right or left vagus. Because of the desperate condition of the patient, I ventured sharp simultaneous pressure over both vagi, encircling the neck with the thumb and index finger of one hand. The pressure was instantly followed by a sudden momentary convulsive seizure of the muscles of the face; the patient seemed dazed for about a minute; at the same time normal heart rate was resumed. There was no return of the tachycardia. Convalescence was uninterrupted, cyanosis and dyspnoea slowly disappeared. About two weeks later, I took an electrocardiogram and fluoroscoped the patient's chest. Nothing abnormal was found. The heart rhythm and sounds were normal. The cardiac response to exercise was normal. One month later I again saw the patient in another typical paroxysmal seizure which had lasted one hour. Again pressure over the right and left vagus alternately had no effect. I therefore again tried simultaneous pressure over both vagi, but this time instead of pressing sharply and suddenly, I slowly brought digital pressure to bear. The attack was again immediately controlled, but without any alarming symptoms; there were no convulsive seizures or a dazed sensorium. Simultaneous double vagus pressure has its risks, but I think its trial is justified in desperate cases especially if the digital pressure be gently graded instead of being sudden and sharp, which is the proper procedure in single vagus pressure.

As an alternative to unsuccessful vagus pressure, pressure on the right or left, or both eye-balls may be tried in attempting to control the tachycardia by inducing the oculo-cardiac reflex, a nerve arc which also induces vagus inhibition. The procedure however, is more painful than vagus pressure and is not as apt to be followed by success. Inducing emesis may also effect the vagus control, hence this may be of value in stopping the attack. Hot water, mustard, turpeth mineral are safe emetics that can be employed. Strophanthin may occasionally be employed hyperdermically or intravenously because of its quick, powerful effect in increasing vagus inhibition. As an addition to any drug or proceedure, morphine hyperdermically, bromides or luminal are excellent adjuvatants, where indicated, in order to quiet the patient's nervous system.

Atropine in small doses three times daily before meals, and an ant-acid powder after meals, with limitation of the diet to bland food eaten slowly, are often of value in avoiding future attacks of paroxysmal tachycardia of gastric or intestinal origin. The powder which I use contains equal parts of magnesium usta, bicarbonate of soda and oleosaccharated peppermint

powder in equal parts; of this the patient is given about ½ teaspoonful three times daily after meals.

**Auricular Flutter** (Chapter X).—This arrhythmia is most frequent in older people with cardiosclerosis during the period of decompensation. It may also occur during the course of acute endocarditis without decompensation. Occasionally auricular flutter is of functional origin. When flutter accompanies acute endocarditis, digitalis does not control the arrhythmia. Since the drug has not been employed in flutter of functional nature, nothing is known of its effect in such cases. In auricular flutter with decompensation and cardiosclerosis, digitalis is of very great value. It should be administered in large doses (Chapter XX) so as to obtain the maximum effects as soon as possible. Digitalis then usually changes the flutter to fibrillation. If then the drug be discontinued, the rhythm often returns to the normal and, coincidentally, compensation is restored. In some patients, several courses of digitalis have been given when flutter recurred, and each time normal rhythm was resumed after the flutter first changed to fibrillation under the influence of the drug.

**Ventricular Escape—Independent Ventricular Activity.**—In order to emphasize the etiological factors involved in this arrhythmia a brief report of a case of this interesting cardiac irregularity with comment on the clinical aspects is given.

S. P., male, aged 20, was first observed on February 4, 1913. He had measles when 3 years old and typhoid when 10; otherwise there was no history of previous illness. He was not addicted to tea, coffee, tobacco or alcohol. Five days prior to hospital admission, he developed a typical attack of acute articular rheumatism involving the ankles, wrists, knees and elbows. The attack was accompanied by fever; there were no chills or gastric disturbances.

Except for swelling and redness of the inflamed joints, the general and neurological examination revealed nothing abnormal. There was no urethral discharge. The complement-fixation test for gonorrhea was negative. The cardiac outline was normal to percussion, the beat was in the fifth interspace, 8.5 c.m. from the midsternal line; the heart sounds were normal; the pulse was rhythmical. The systolic and diastolic blood pressures were within normal limits. The temperature ranged between 101° and 103°. There was no dyspnoea. The patient did not appear very ill. Sodium salicylate in moderate doses was given for two days.

Two days after hospital admission, a transient pulse irregularity appeared. Six days thereafter, it recurred and clinically resembled extrasystoles; no tracings were made at that time. February 12, the irregularity occurred with every third or fourth beat. From that day frequent polygraphic and later, electrocardiographic tracings were taken. February 14, a rough blowing systolic murmur was heard at the apex for the first time. Occasionally there were runs of from three to twelve stronger thumping beats

unaccompanied by the murmur; studies of the tracings showed that these were due to simultaneous action of auricle and ventricle, and that the ventricle was beating automatically and independently (ventricular escape, Chapter X). Four days later the arrhythmia appeared only infrequently and the systolic murmur had almost entirely disappeared. The patient left the hospital feeling well.

As possible causes for the production of automatic ventricular action, neurogenic, toxic and organic factors require consideration. A neurogenic factor in the sense of a so-called neurosis due to extracardial conditions (for example, gastric disorders) causing abnormal excitation in the afferent (centipetal) arm of a reflex arc, can be dismissed in our case because of the type of the disease, its course, and the definite completion of the arrhythmia with the end of the rheumatic attack. Concerning toxins in general, their action seems to depend upon their complicated chemical composition and upon intricate chemical reactions taking place in the body. By analogy, it seems possible that a rheumatic toxin may also produce an arrhythmia by directly poisoning the heart, although there is no evidence for such assumption in this case. Concerning an organic cause for the arrhythmia, it is to be recalled that the patient developed a loud systolic murmur at the apex one week after the appearance of the arrhythmia; the murmur remained for two days, then gradually disappeared. It also disappeared during those arrhythmic beats when auricle and ventricle contracted simultaneously, an apparent corroboration that it was due to mitral insufficiency, organic or relative in nature. It is not my intention to discuss cardiac murmurs at any length in this connection (q.v. Chapter XIII). Briefly, systolic apical murmurs which occur during the course of any febrile disease, and then disappear without evidence of an organic cardiac lesion, are by no means infrequent. On the other hand, organic murmurs usually increase in intensity and do not disappear. In the case under discussion, the occurrence of the murmur in conjunction with acute articular rheumatism makes its presence suspicious of some transient valvular or myocardial involvement. Rheumatic infections cause myocardial inflammation in the form of submiliary myocardial (Aschoff) bodies (Chapter IV). Thus, healed or healing isolated Aschoff bodies have been found on the interventricular septum in hearts which were the subjects of rheumatic reinfection. During their inflammatory state, if situated close to, or even partly involving the bundle of His before its division, they can conceivably cause local irritation and excitation sufficient to produce occasional ventricular automatism, with beats of supraventricular origin. The bundle, however, need not be sufficiently compromised to prevent the excitation impulse from following its normal course in the conduction system—a fact which probably accounts for the identical electrocardiographic complexes here present in all the beats, both rhythmic and arrhythmic.

Right and left vagus pressure had no effect on auriculo-ventricular sequence. One of the atropine experiments was followed by a number of

independent ventricular contractions, with no marked difference between ventricular and auricular rates. This observation does not necessarily exclude the possibility of an organic cause for the ventricular "escape," for an irritative lesion which does not entirely and permanently affect the bundle may nonetheless upset normal nerve control and mechanism, and make the latter susceptible to atropine poisoning. It would thus seem that the automatic ventricular mechanism was not sufficiently sensitive to respond to vagus pressure, but that atropine poisoning prevented inhibitory vagus control and permitted ventricular escape.

Summarized, a case of independent ventricular activity is described: The lowest ventricular rate is 50 per minute, the usual rate is 60 and remains so whether independent ventricular activity is present or not. The electrocardiographic complexes of all beats are identical. At one time atropine administration was followed by ventricular escape. The occurrence of automatic ventricular activity during the course of acute articular rheumatism and its disappearance later, combined with a study of the physical signs make it probable that a small transient myocardial inflammatory focus (Aschoff body) at or near the auriculo-ventricular bundle is the irritable cause of the abnormal mechanism. Therefore it may be stated that transient independent ventricular activity (ventricular "escape") may occur with no change in the usual path followed by the excitation impulse, with no difference of rate between normal and abnormal beats, and with no marked retardation of the auricular rate.

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## CHAPTER XII

## TELEROENTGENOGRAPHY, ORTHODIASCOPY AND FLUOROSCOPY

Orthodiascopy is essentially a method of outlining a viscus by means of parallel roentgenographic rays; in this manner the exact size of the organ is reproduced. With the tube stationary and near the patient, the rays from the focus of the tube diverge; hence the object to be examined becomes artificially enlarged to a variable extent. If the organ itself is large, this distortion is magnified, for the impinging rays become still more divergent; an hypertrophied heart, for example, produces a disproportionately enlarged shadow. This was well exemplified in a case in which the roentgenogram taken two days before death showed the shadow of a tremendously enlarged heart. At necropsy the heart was found only moderately enlarged; pericarditis causing a tightly adherent pericardium prevented terminal cardiac dilatation which might have been assumed as the cause for the enlarged roentgenographic shadow.

Two methods of avoiding distorted images have been devised. (1) Teleroentgenography and (2) orthodiascopy.

**1. Teleroentgenography.**—In following this procedure, the X-ray tube is placed about six and one half feet (2.3 meters) from the patient, so that the rays reach the organ approximately parallel. The X-ray plate should be held parallel to the anterior surface of the midsternum, and the X-ray tube so placed that its central rays are at right angles to the center of the plate at about the level of the infrasternal notch. Properly applied, teleroentgenography has given excellent results. Its great advantage is its rapid technic under modern conditions. There is also no danger of an X-ray burn to the operator. Compared with orthodiascopy, its disadvantages are the amount of X-ray plates required and especially the fact that the action of the heart cannot be actually studied and observed, for a photograph can naturally give only the picture of the heart at and during the time the plate is exposed. Another advantage of orthodiascopy over X-ray plates is that questionable shadows may be at once interpreted without the necessity of having the patient return for another plate. As will be shown later, very many important facts can be gleaned by observing the heart in action.

**2. Orthodiascopy.**—With this method, the focus of the X-ray tube and the center of the fluorescent screen are adjusted so that they are in a straight line, and the X-ray tube and the fluoroscopic screen move together. As the outline of a viscus (in this case, the heart) is seen, the screen is moved and

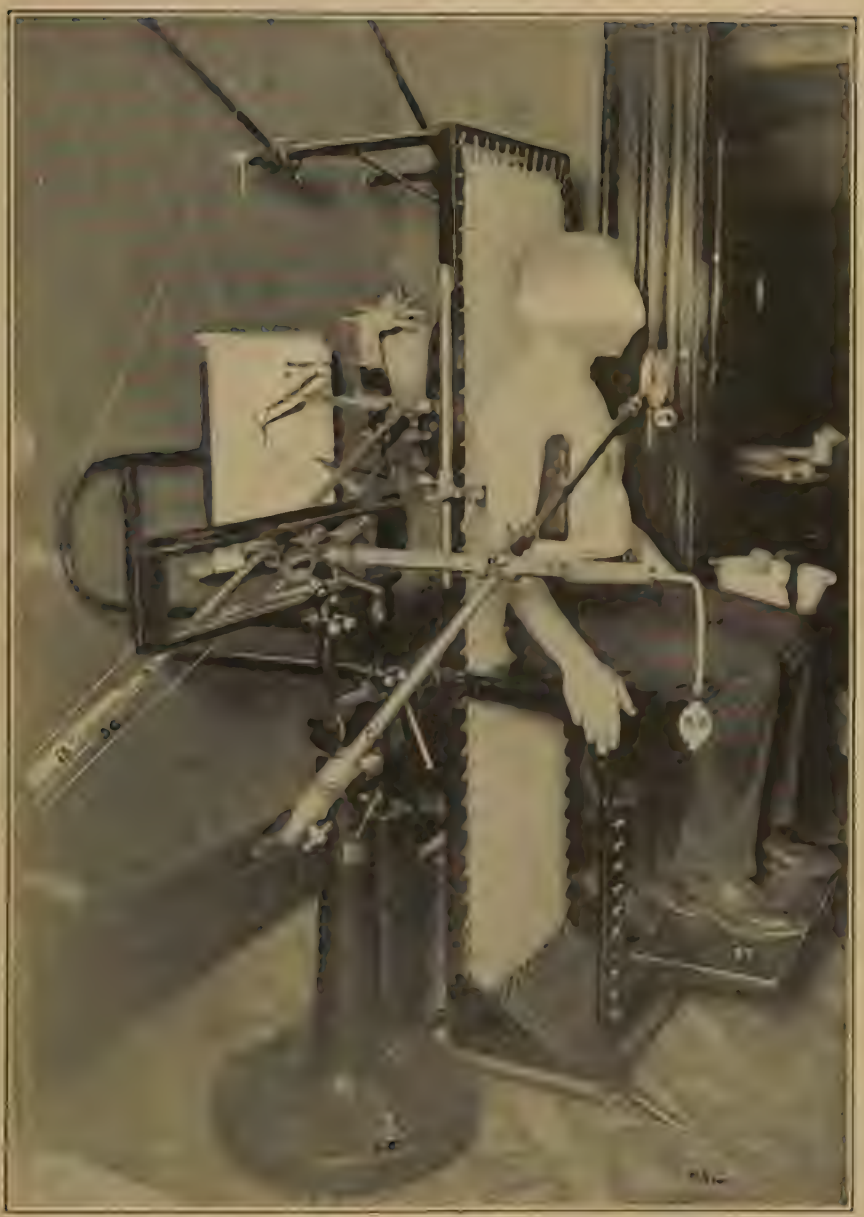


FIG. 234.—Groedel orthodiascope, front view. The patient sits with his feet resting comfortably on the adjustable step (ST); the side plates (SP) can be lowered or raised and pushed in or out; they are placed so that they fit snugly in the axillæ and against the chest of the patient. The fluoroscopic screen (FS) is grasped by the finger of the right hand in a small ring (R). The bar B′ carrying the screen is connected by the hollow bar (CB) with the bar B″; the latter carries the X-ray tube so that any motion imparted to the fluoroscopic screen by the observer moves the X-ray tube as well. By loosening the nut (N) the bar (B′) may be slid along the crossbar, thus the screen may be placed at any convenient distance from the patients chest. RB, rubber bulb used for marking purposes; SC, sliding counterweights so that the screen and X-ray bars may be properly counterpoised; C, cranks used to place the table in the horizontal position if necessary; LS, levelling screws; FSW, the electrical foot switch.

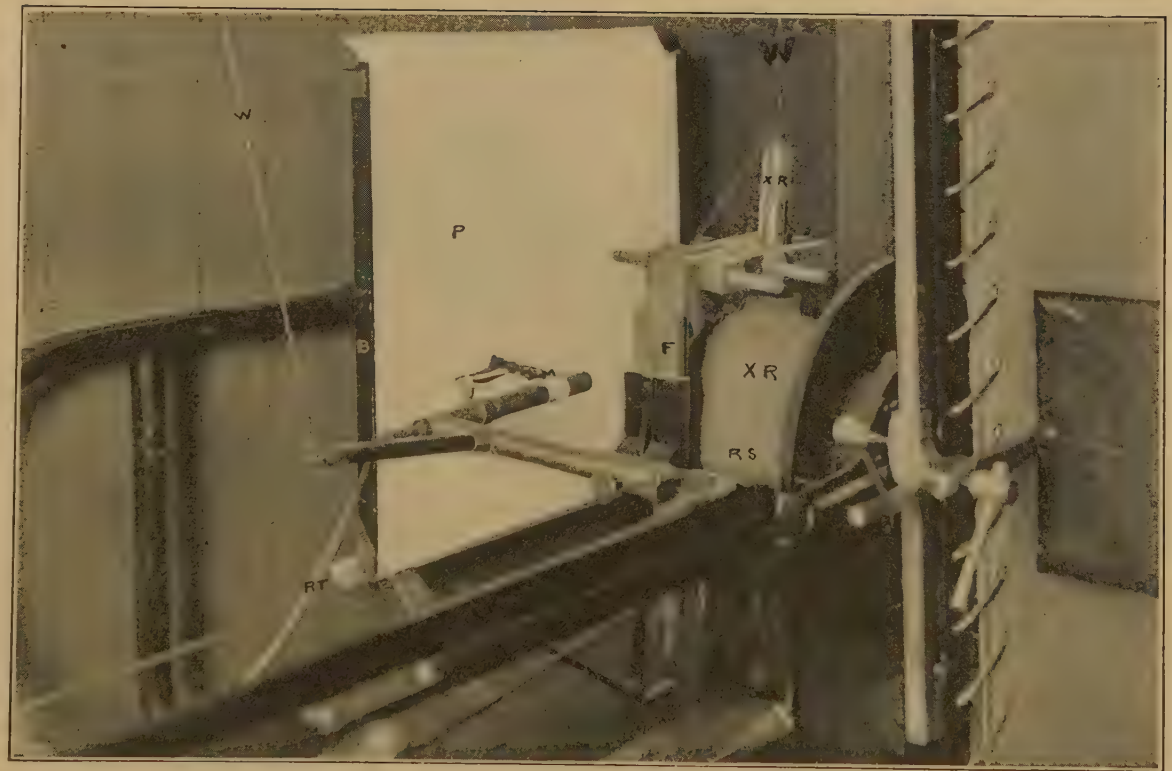



FIG. 235. Details of marking mechanism: XR, X-ray tube which may be raised or lowered in its frame (F). It is partially protected by its rubber shield (RS). The marker (M) is in line with the center of the screen (see Fig. 234) and the focus of the X-ray tube. The rubber tubing (RT) is connected with the rubber bulb (Fig. 234) through the hollow cross bar. When the bulb is squeezed and the marker inked, the pencil is pushed forward and dots the paper (P) fastened upon the board (B). W, are the wires connecting the X-ray tube with the coil.

brought in alignment, so that, with the simultaneous movement of the tube and screen, parallel and not divergent rays impinge upon the organ and reach the observer. The application of this principal is exemplified in the Groedel apparatus (Figs. 234, 235). It is the one I have found suitable for orthodiascopy of the heart. Examinations can be made in the sitting, standing, or lying postures. For general convenience as well as for the comfort of the patient I prefer the sitting position. After the patient is properly seated (see legend, Fig. 234) the room darkened, and the electric foot switch pressed upon, a rapid survey is first taken of the entire heart by moving the fluoroscopic screen over the cardiac area. The cardiac outline is thereafter systematically mapped out. The central spot of the screen (F. S. Fig. 234) is brought over the edge of the area to be delimited; this point is then marked by pressing the rubber bulb with the left hand. My practice is to start the tracing on the left side by dotting out the left outer limit of the aortic arch, then continuing downward and marking the other curves of the heart (Fig. 236) until the entire left side is outlined. To expose the lowest part of the apex, the patient is asked to hold his breath for a moment at the end of a deep inspiration. The upper right border of the aorta, then the remainder of the right border of the heart, are mapped out down to the diaphragm. Finally, the diaphragm is outlined on each side. The cardiac contour is best dotted out during the time of systolic contraction. There is very little danger of X-ray burns since the amount of current used is small (about 5 amperes). The entire orthodiascopic procedure requires generally about two minutes. During the examination and for a minute or two thereafter, the observer can carefully study the heart action, and thus not only gain important data regarding the various types of contractility, especially of the left ventricle and aorta, but also fluoroscopically study shadows, the interpretation of which may be uncertain in a roentgenogram.

The details of the marking mechanism are shown in Fig. 235.

**Orthodiascopic Tracing of the Normal Heart.**—It is important to have a definite visualized image of what the heart actually resembles when seen in the chest, for it will aid tremendously in checking up some of the percussion findings at the bedside where naturally an X-ray is not at hand. The combined contour of the normal heart and aorta is an irregular oblique ovoid; its larger end is directed downward and to the left (Fig. 236). The outline is made up of several curves formed by different portions of the heart. On the right side above, we find the ascending aorta, which forms a curve with a slight outward convexity ($A^1$, Fig. 236). Above it, the great vessels are sometimes seen as indefinite shadows. Very rarely, the superior vena cava is visible as an attenuated shadow stretching across the aorta. Below the ascending aorta is the curve formed by the right auricle (R. A. Fig. 236). It is usually a well-defined arc and forms an acute angle with the arch of the diaphragm. In cases of extreme right ventricular enlargement, or in pericarditis with effusion, this angle may be obliterated. The left-sided

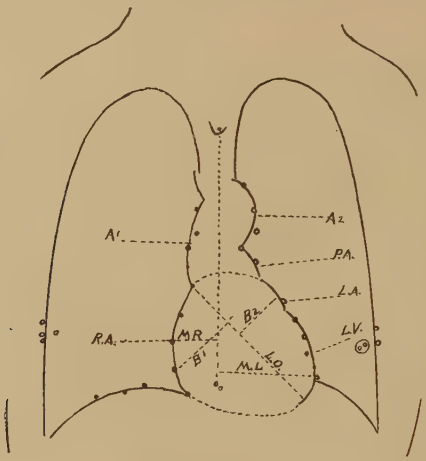



FIG. 236A.

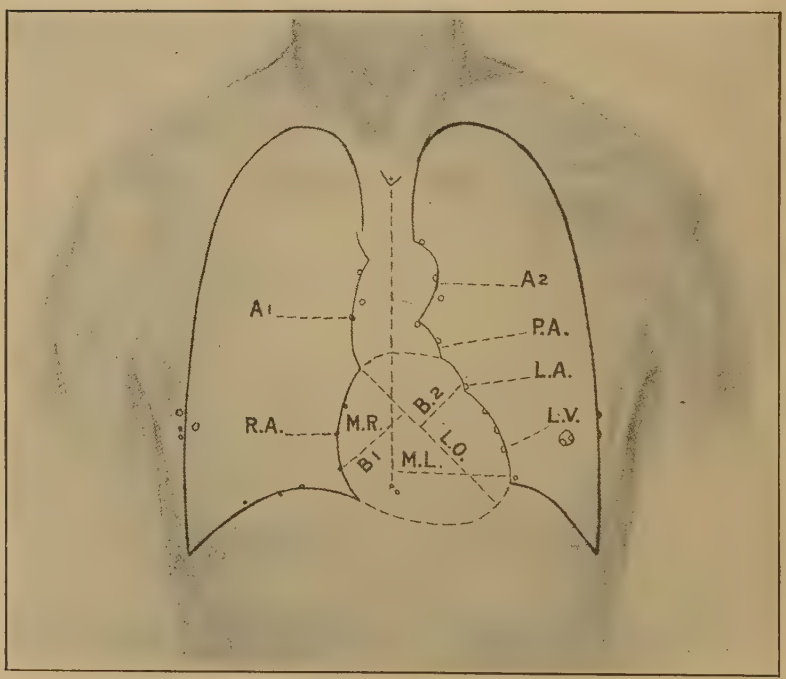



FIG. 236B.

FIGS. 236A and B.—Orthodiascopic tracing of the normal heart.[1]

AI, Ascending aorta; R.A., right auricle; $A_2$, aortic arch; P.A., pulmonary artery; L.A., left auricular appendix; L.V., left ventricle; M.R., greatest distance from median line to the right; M.L., greatest distance from median line to the left; L.O., longest oblique diameter of the cardiac ovoid; $B_1$–$B_2$, greatest width of the orthodiascopic tracing.

[1] In order to show clearly the areas occupied by the cardiac silhouette, the orthodiascopic tracings have been blocked in figures of the chest.

silhouette is composed of four more or less distinct curves. From above downwards, these are the aortic arch ($A^2$, Fig. 236), the pulmonary artery (P. A.), the left auricular appendix and auricle (L. A.), and the left ventricle (L. V.). The arch of the aorta protrudes slightly beyond the left sternal border, describing a convex arc; when the aorta curves sharply downward and backward to reach the spine, this arc assumes a knoblike prominence. The normal pulsatile excursions are slight (about 0.5 centimeter) and require careful scrutiny in order to identify them. The average length of the arch, visible as a separate shadow, is from 3 to 5 centimeters. The left margin of the descending aorta can sometimes be traced downward for several centimeters as a lighter shadow; in exceptional instances, it is dimly seen as a pulsating silhouette behind the body of the left ventricle. Beneath the aortic arch and distinguished from it as a much smaller and less convex silhouette is the pulmonary artery; its average visible length is from 2 to 3 centimeters. The contour of the pulmonary artery is distinguished from that of the underlying left auricle by the difference in pulsation time. The left auricular curve is obtusely oblique and variable in size; it consists mainly of the left auricular appendix, but sometimes also includes part of the left auricle. The left ventricular curve constitutes the greater portion of the left ventricle, and forms its ovoid end. The extent to which the left ventricle is visible depends upon the shape of the heart and the mobility of the diaphragm. In stout individuals with thick abdominal walls, diaphragmatic excursion is usually limited, and the apex of the heart may not be visible; in thin individuals with good diaphragmatic mobility the entire left ventricular surface including the apex is plainly seen at the end of inspiration.

In addition to giving definite information about the exact size of the heart, orthodiascopy has several advantages over teleroentgenographic plates. For example, as not infrequently happens, there may be a dubious shadow which in a plate may be variously interpreted as a mediastinal tumor or an aortic aneurism. Orthodiascopy can at once clear up this distinction by seeing the actual pulsatile excursion of the aorta in suspected aneurism, and by turning the patient in various lateral directions in order to discover whether a suspected mediastinal tumor fills out the posterior mediastinal space (Chapter XVI). Besides, orthodiascopy gives unique and important information regarding the type of contraction of aorta and pulmonary artery, and of right auricle and left ventricle. It is direct observation of the contraction of the left ventricle which is of great help in diagnosing questionable hypertrophy, as well as in segregating individuals who complain of "weak heart" (Chapter XVIII) but who none-the-less show vigorous ventricular contractions. The manner and amount of aortic pulsatile excursions sometimes helps in the diagnosis of aortic regurgitation where that diagnosis may be otherwise uncertain (Chapter XV). Orthodiascopy can also demonstrate extrasystoles and the irregular ventricular action typical of auricular fibrilla-

tion, as well as other data of interest found in various types of cardiac irregularities.

**Orthodiascopic Standards.**—In order to determine accurately the standard size of the heart, numerous statistical studies and measurements of orthodiascopic tracings have been made. For this purpose, oblique and other diameters of the cardiac ovoid (Fig. 236) have been devised and measured; from these, attempts have been made to establish normal cardiac areas. The diameter representing the greatest width of the heart ( B 1 B 2, Fig. 236) depends for its accuracy upon the left auricular curve; this is a variable quantity even in normal individuals and hence unreliable as a standard for measurement. Some observers have, therefore, chosen the largest oblique diameter (L.O., Fig. 236) and the distance of the cardiac borders from the median line (M.R., M.L.) as being more exact. These axes have been studied principally by Groedel, Dietlin, and Veith in individuals of both sexes, of various ages, weights, and size, with varying types of thorax, and following different vocations. The chief objection to the use of the long axis (L.O.) as a standard is the fact that it is by no means always possible to see and delimit the tip of the apex, partly because some patients do not breathe properly, partly because in some individuals diaphragmatic excursions is limited, and often, (especially in those with heart disease) dyspnoea interferes with accurate X-ray exposure of this area. All these considerations may considerably influence the value of the long axis as an accurate standard of measurement. Since the transverse diameter is not so affected, the most recent observations are based upon this as the best available basis of measurement. Morever it has been found as accurate as any of the other measurements, including estimation of the cardiac area. Groedel has found the following average figures in the vertical orthodiagram:

| | M.R. (see Fig. 236A and B), c.m. | M.L., c.m. | L.O., c.m. |
|---|---|---|---|
| Adult males | 4.6 | 8.4 | 14.0 |
| Youths | 4.1 | 7.8 | 12.7 |
| Adult females | 3.9 | 8.0 | 12.9 |
| Young females | 3.7 | 7.2 | 12.1 |

Tables of measurements of other observers are given at the end of this chapter. One of these is based upon the teleroentgenographic examination of soldiers (A. E. Cohn). Since, however, the size of soldiers hearts, even after several months of active service, has not been found to vary appreciably from that of the civilian adult of the same weight and physique, these tables may also be used as a standard of size of the adult male heart. In this connection it is interesting to note that another observer (Bertrand

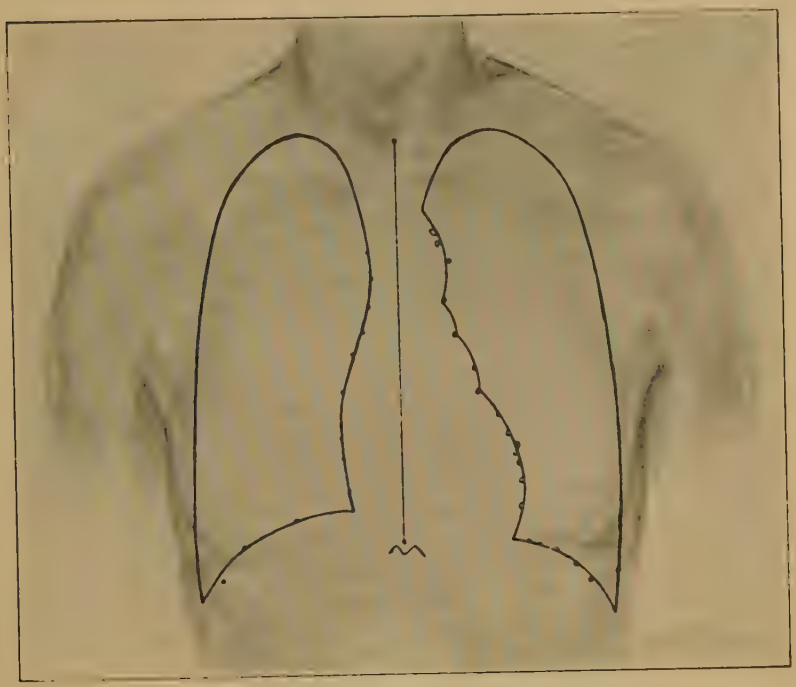


FIG. 237.—Orthodiascopic tracing of long and slender heart.

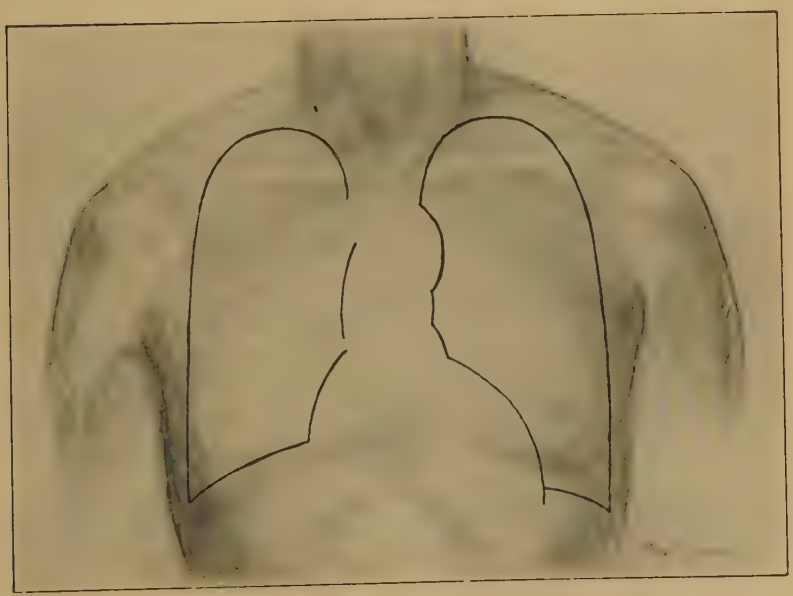


FIG. 238.—Copy of orthodiascopic tracing of a broad heart.

Smith) making similar teleroentgengraphic studies of the "irritable heart of soldiers" (Chapter XVIII), has found that these likewise do not vary from similar corresponding adult hearts in the civilian.

In all these tabulated measurements, a certain correlation has been found to exist between the cardiac area, and size and weight of the individual. The ratio of the heart to body weight in the adult male is approximately 1:200. There are important considerations, however, which tend to lessen the value of measurements as standards. Size, weight, type of thorax, and occupation are factors which have already been mentioned. In these alone there is a wide range of maximal and minimal measurements which may amount to more than 3 c.m. Another factor capable of producing wide variations from the standard and still be within the normal is that of different types of cardiac contour which do not correspond with the muscular make-up of the individual. The extremes are those with long, slender, graceful hearts scarcely resting upon the diaphragm, and those whose hearts are broad, lying flat almost along their entire lengths. For example: In a youth of 19, tall and slender (Fig. 237) M.R. = 3 c.m.; M.L. = 6 c.m.; the other is the orthodiagram of a robust male of 35 (Fig. 238). Both are healthy and have normal hearts; marked differences in all diameters are immediately apparent by reference to the diagrams. An added consideration, already alluded to, tending to lessen the value of measurements as comparative standards is the difference produced by movements of the diaphragm. When excursion of the latter is limited, less of the heart is uncovered, its ovoid contour appears flattened, and the transverse measurement is correspondingly enlarged. With a mobile diaphragm the reverse is usually true. From all these facts it is apparently impossible to establish normal mathematical orthodiascopic standards of measurement. This statement, however, does not preclude such general conclusions as that "normal" hearts may be unusually large or small, or that pathologically enlarged hearts often overstep the somewhat ill-defined normal limits. There is naturally no doubt of the value of orthodiascopy in the determination of marked variations in the size of the heart.

**The Narrow and Broad Heart.**—Returning to the discussion of the two types of cardiac silhouette (Figs. 237, 238)—the abnormally narrow and the abnormally broad—there is not only a difference in size but also a marked difference in form. The left ventricle of the broad heart is flat, it hugs the diaphragm, and forms a flattened ovoid. The other type is ellipsoid, and rests lightly upon the diaphragm; Figs. 239, 240 are additional examples. One silhouette is that of a man of 25 suffering from bronchial asthma (Fig. 239); the other that of a woman of 26 suffering from marked vasomotor symptoms (Fig. 240). This type, sometimes called the "drop heart," has assumed considerable clinical importance, for it has been found in patients with visceroptosis; it is regarded as one of the characteristics of habitus asthenicus, and is apparently the type of cardiac hypoplasia described in stasus thymo-lymphatious. The symptoms I have found in these individuals are those

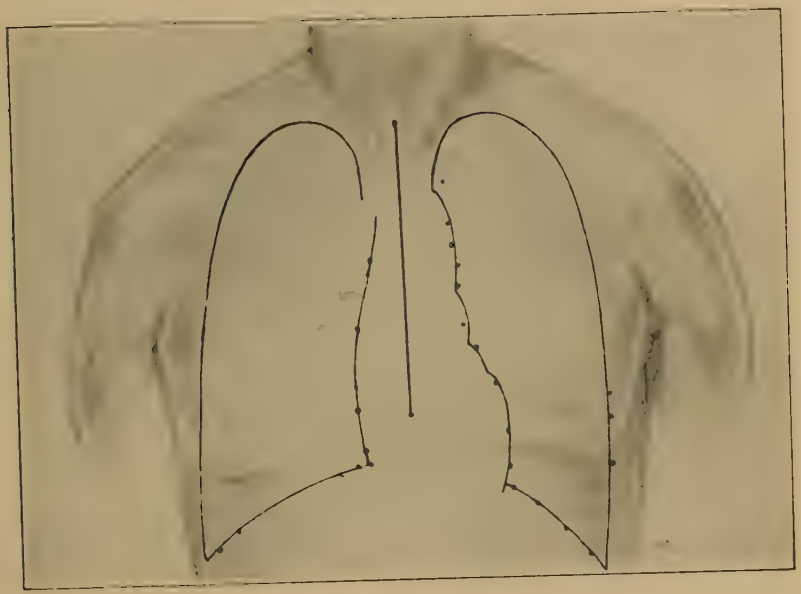


FIG. 239.

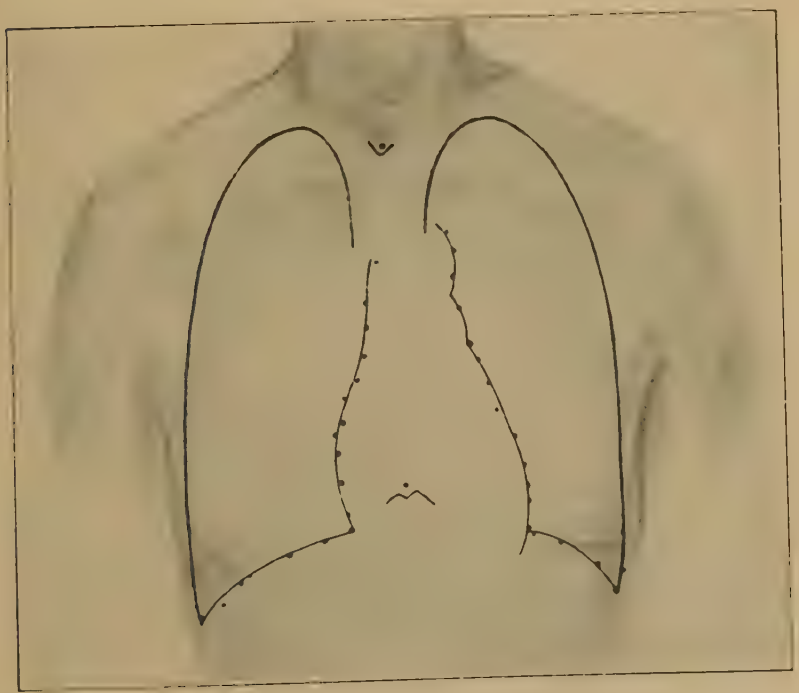


FIG. 240.

FIGS. 239, 240.—Orthodiascopic tracings of abnormally slender hearts resting lightly upon the diaphragm ("drop" hearts).

FIG. 241.—Orthodiascopic tracing of a woman of 30 with a compensated mitral regurgitant lesion showing normal cardiac contour and size.

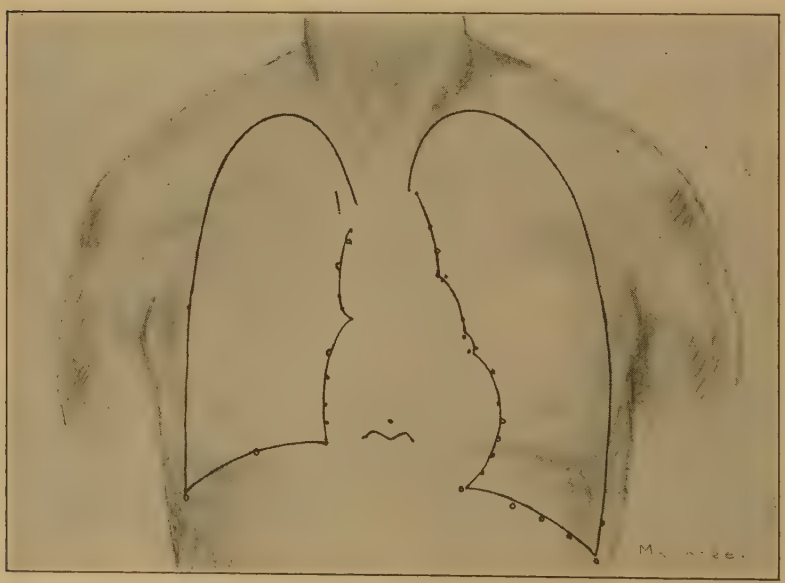


FIG. 242.—Orthodiagram of a girl of 17 with a compensated double mitral lesion. The tracing is normal in size and contour.

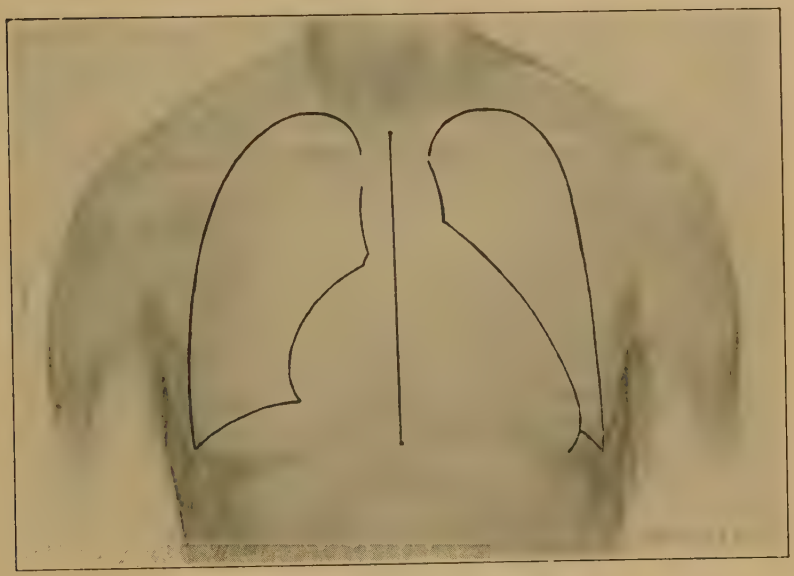


FIG. 243.

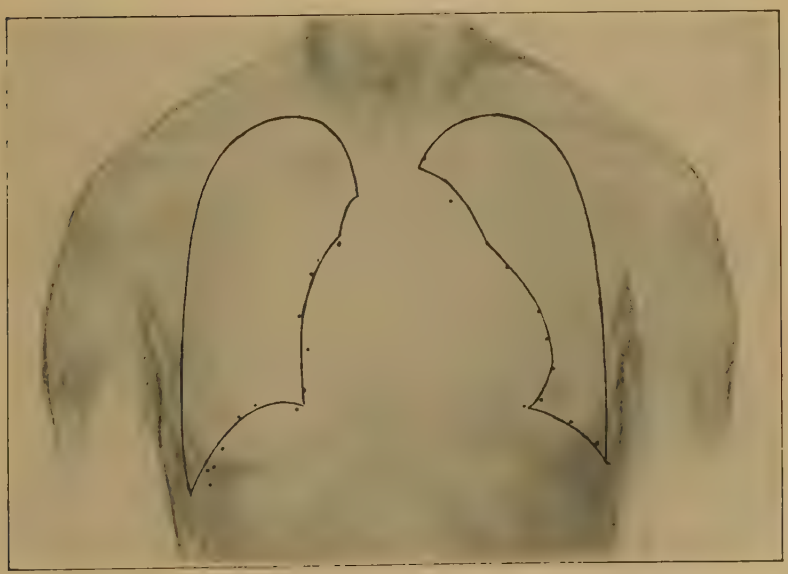


FIG. 244.

FIG. 245.

FIGS. 243, 244, 245.—Examples of globular hearts of varying degrees of rotundity and size. From three cases of decompensated double mitral lesions with auricular fibrillation. In Fig. 243 the aortic curve is normal.

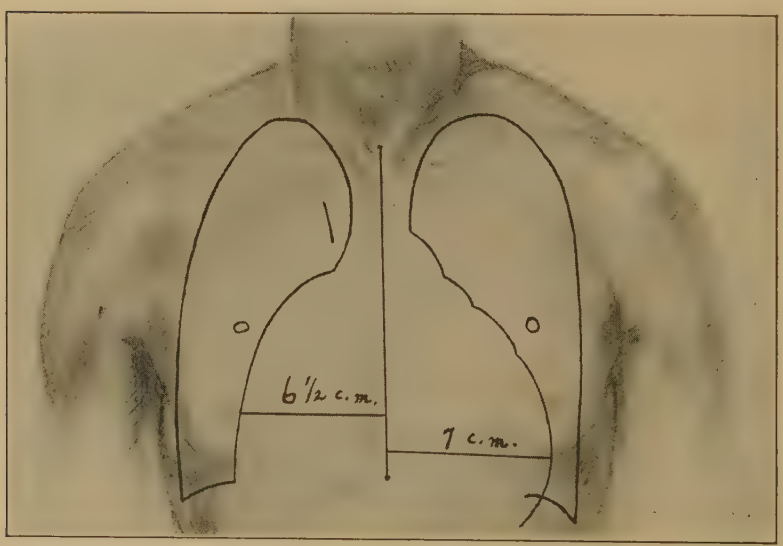



FIG. 246.—Globular heart of extreme size. Copy of an orthodiascopic tracing of a boy of 11 suffering from a double mitral lesion and auricular fibrillation.

connected with vasomotor instability—flushed, pale and cold extremities, dizziness, etc. These are often erroneously interpreted as due to a "weak heart" (Chapter XVIII). It should be emphasized however that individuals with habitus asthenicus as well as those with vasomotor symptoms often have hearts of perfectly normal size and shape.

**Orthodiascopy in Mitral Disease.**—Orthodiascopy has been used as a method of establishing the diagnosis of valvular lesions upon the assumption that these produce typical changes in cardiac contour. With reference to mitral disease, it is true that many advanced decompensated patients conform in a general way to an orthodiascopic type, but those with quiescent and compensated lesions may possess hearts of normal size and contour. This indeed might be suspected from postmortem findings of mitral disease where the process has caused little change from the normal in the size, form and weight of the organ. Two illustrations are given in Figs. 241 and 242 of mitral disease with a normal sized heart. The former is of a woman of 30 with a mitral regurgitant lesion of ten years duration; the latter is from a girl, aged 17, with a double mitral lesion of four years duration. In both there were typical physical signs of the respective valvular lesions; both were compensated, the patient with double mitral disease was still suffering from rheumatic manifestations at the time that the orthodiagram was taken. Another instance is the tracing of a tall, well-built lad of 18, (Fig. 251), who recently recovered from the acute manifestations of mild rheumatic mitral regurgitation.

Decompensated double mitral lesions with auricular fibrillation are those which usually conform to a general orthodiascopic type. The aorta may retain its normal outline or be dilated to a varying degree, even in the absence of physical signs of an aortic lesion. The remainder of the cardiac contour forms one large fused outline—the globular heart—consisting of the dilated pulmonary artery, left ventricular and auricular curves on the left side, and a ballooning out of the right auricular curve on the other. Figures 243, 244, 245 are examples showing varying degrees of size and rotundity of outline from three cases of decompensated double mitral lesions with auricular fibrillation. All the patients were adults. In one (Fig. 243), the aortic curve is normal; in the others, it is dilated. Another interesting example of the globular heart (Fig. 246) is the orthodiagram of a boy of 11 with a decompensated double mitral lesion and auricular fibrillation. Occasionally, the orthodiascopic tracings of patients with these lesions show neither extreme enlargements nor definite rounded outline. Thus Fig. 244 is the tracing of a man of 50 who only recently developed auricular fibrillation; Fig. 248 is that of a woman of 46 who has had the valvular lesion and arrhythmia for many years. Other variations from the typical globular form are sometimes found in chronic cases of double mitral disease with fibrillation in which there is disproportionate right- and left-sided enlargement. For example, the tracing in Fig. 250 is from a patient of 55 with an old double

FIG. 247.

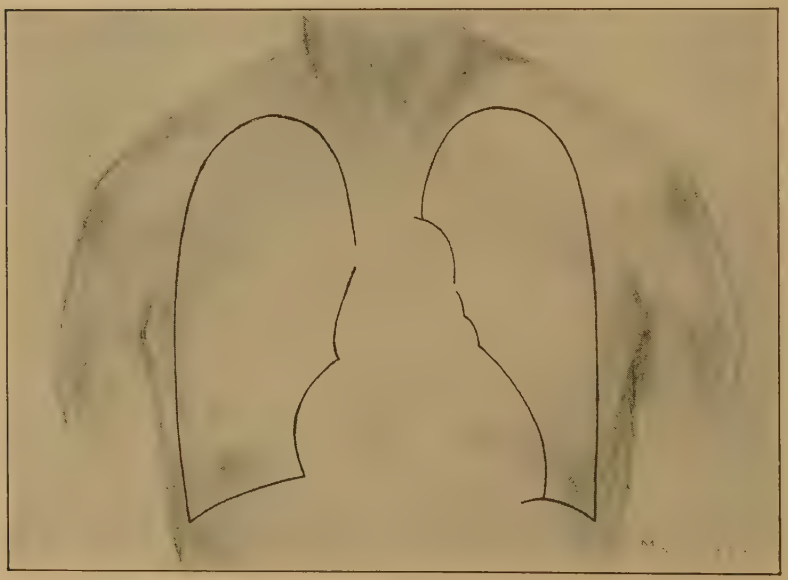


FIG. 248.

FIG. 247, 248.—Orthodiascopic tracings showing a somewhat globular heart with moderate enlargement. From cases with double mitral lesions and auricular fibrillation.

FIG. 249.—Extreme rounded right-sided enlargement. The left contour is enlarged downward. From a case of double mitral lesion, auricular fibrillation and cardiac failure.

FIG. 250.—Copy of orthodiascopic tracing of a woman of 35 with a double mitral lesion of 20 years' duration. There is only very slight left-sided enlargement.

FIG. 251.—Normal orthodiascopic tracing of a tall boy of 18 suffering from a recent mitral regurgitant lesion.

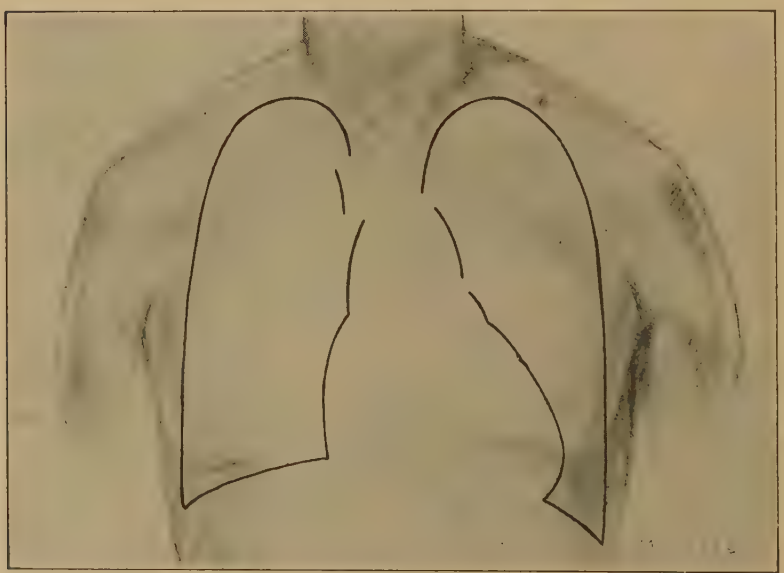


FIG. 252.—Orthodiagram of a woman of 35 with a mitral regurgitant lesion showing a somewhat ovoid contour with moderate dilatation to the left.

mitral lesion, frequently decompensated, and with auricular fibrillation. There is a sharply rounded right-sided enlargement, the left side is enlarged downwards and outwards; the resultant silhouette is an irregular ovoid.

Of great interest from the orthodiascopic standpoint are those patients with chronic double lesions without auricular fibrillation in whom there has never been any severe break of compensation. The tracings are sometimes normal in configuration and size, as in the cases already described (Figs. 241, 242), or the enlargement is too slight to be of diagnostic significance. Thus, a woman of 45 had a double mitral lesion over twenty years. Recently she developed occasional extrasystoles accompanied by a subjective feeling of "weakness" in the chest. Otherwise she has never had any cardiac complaint. The orthodiascopic tracing (Fig. 251) shows only very slight left-sided enlargement.

In chronic mitral regurgitation with decompensation, the orthodiascopic tracing is usually ovoid or somewhat circular in shape. The longest axis lies diagonally, the larger end corresponding to the left ventricle. The elements constituting the abnormal form are moderate enlargement of the left auricular, the left ventricular, and the right auricular curves. An instance of this is seen in Fig. 252, a female aged 35, with a rheumatic mitral regurgitant lesion; the orthodiagram shows an ovoid heart with moderate dilatation of the left border. If decompensation in patients with chronic mitral regurgitant lesions is extreme, the entire contour becomes circular.

**Orthodiascopy in Rheumatic Aortic Disease.**—With reference to rheumatic lesions of the aorta, one of the chief fluoroscopic characteristics is the degree of the aortic excursion—the aortic fling. This is often observed in patients with double aortic lesions whether compensated or not. As a rule, the aorta itself is not permanently enlarged, although the erroneous impression is gained from its occasional violent systolic expansion. There appears to be some relation between the clinical severity of the disease as gauged by the dyspnoea and cardiac reserve power, and the degree of the aortic fling. In patients severely ill, the entire aorta—the ascending arch and the descending portions—pulsates so violently as to resemble an aneurism; those less ill usually show excursion limited to individual sections of the aorta. Another fluoroscopic characteristic of aortic lesions is the sharp, vigorous left ventricular contraction, with a distinct lifting motion of the apical region. When left ventricular hypertrophy is extreme, the contraction of the apical region is so marked as to appear separate from the remainder of the left ventricle, the whole giving the impression of a see-saw motion. In general, it may be stated that the ventricular excursion during systole is greater in aortic valvular lesions than in the other types of cardiac disease; this is sometimes of diagnostic significance in suspected aortic disease with indistinct and atypical physical and clinical signs.

The change in cardiac contour found in aortic valvular lesions is more constant than in mitral disease. The apical region is enlarged and obtuse,

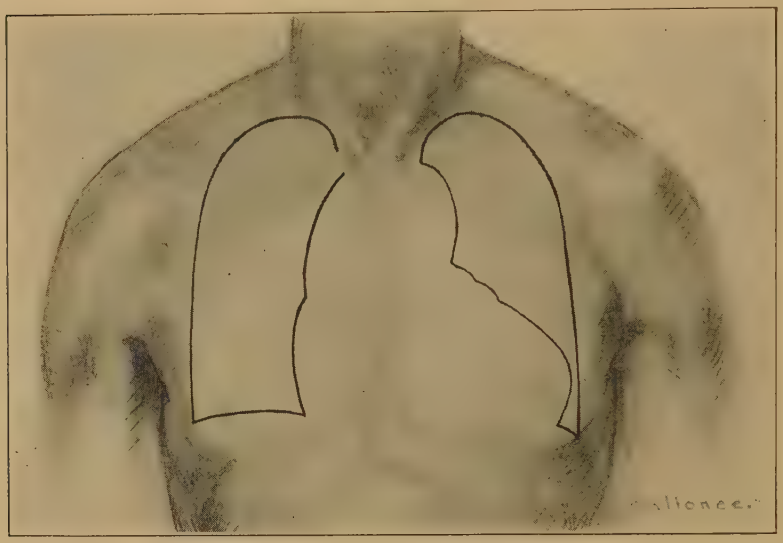


FIG. 253.—Orthodiagram of old double aortic lesion showing left ventricular hypertrophy (so-called "duck-shaped" heart).

FIG. 254.—Orthodiagram of a patient with aortic regurgitation. From a boy of 19, showing left ventricular hypertrophy and moderate enlargement of the aortic arch. The pulmonary and left auricular curves are small.

and the apex itself is often found near the axillary line. The body of the left ventricle is egg-shaped. The right auricular curve is moderately increased in convexity, the entire auriculo-ventricular outline presents an abnormally broadened, flat, somewhat egg-shaped oval, with the wide end at the apex. In conjunction with curves formed by the aorta and pulmonary artery, the entire cardiac outline sometimes roughly resembles the "duck-shaped heart" described by Groedel.

Several typical illustrations of double aortic lesions follow: Fig. 253 is the orthodiagram of a woman of 55 with an old rheumatic history. There was dilatation of the arch and first part of the aorta. The entire left ventricle was greatly hypertrophied, its outline enlarged, the pulmonary curve flattened, the left auricular curve very small. Fluoroscopically, there was very marked aortic fling of the entire visible aorta.

Figure 254 is the tracing of a boy of 19 with a rheumatic aortic lesion of twelve years' duration. There was marked left ventricular hypertrophy, the pulmonary and left auricular curves were small. There was moderate dilatation of the aortic arch; fairly marked aortic fling was present. Whether the right-sided enlargement was due to the right auricle itself, or whether an enlarged left ventricle pushed the right auricle abnormally to the right, was not clear.

While the entire foregoing description applies especially to double aortic lesions, with some difference it also applies to the comparatively rare cases of aortic stenosis alone. In the latter the size and shape are similar to the former, but enlargement of the various curves is not extreme, and the aortic fling is not as marked.

**Orthodiascopy in Combined Aortic and Mitral Disease.**—When both the aortic and mitral valves are affected, the orthodiascopic tracing usually follows the type of the lesion clinically predominant. Thus, Figs. 255 and 256 are taken from two cases with mitral regurgitation and double aortic lesions. The first is that of a girl of 12 in whom endocarditis developed at the age of 5; the second, that of a girl of 19 the duration of whose endocarditis could not be determined. The first is not characteristic of either lesion; the second shows the characteristics of aortic lesions from which this patient clinically suffered.

**Orthodiascopy in Aortitis.**—Aortitis (Chapter XVII) with secondary involvement of the aortic cusps belongs in a different category from the rheumatic valvular lesions. Although calcification and thickening of the aorta do not necessarily imply enlargement and dilatation, nevertheless the latter is the rule in aortitis. The lesion may be confined to the first part, (the ascending aorta), to the arch, or to the descending thoracic aorta; or it may involve the entire thoracic and even part of the abdominal aorta. Hence, it is important to distinguish aneurismal dilatations and enlargements of various parts of this vessel.

FIG. 255.

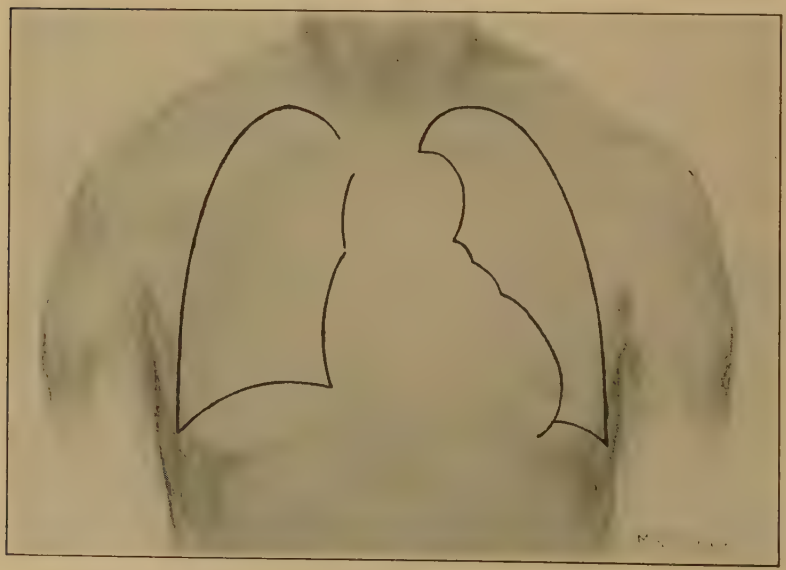


FIG. 256.

FIGS. 255, 256.—Orthodiagrams of two cases of double mitral and aortic lesions. The outlines are characteristic of the latter in Fig. 256. Fig. 255 is not characteristic of either lesion.

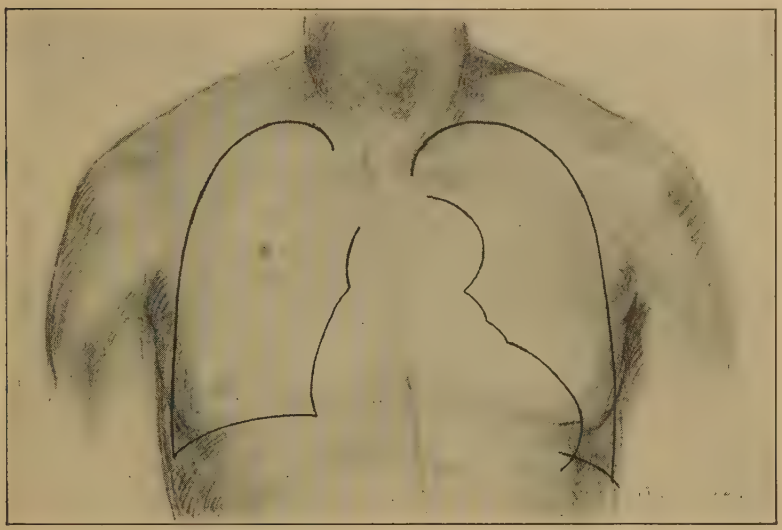


FIG. 257.—Dilatation of the aortic arch and left ventricular hypertrophy. From a patient with chronic nephritis and moderate hypertension.

FIG. 258.—Aortitis with enlargement of the first portion and arch of the aorta, and left ventricular dilatation. From a patient of 65 with symptoms of general arteriosclerosis appearing one year after a severe grippe infection.

FIG. 259.—Dilatation of the aortic arch and left ventricular hypertrophy. From a patient of 60 with arteriosclerosis.

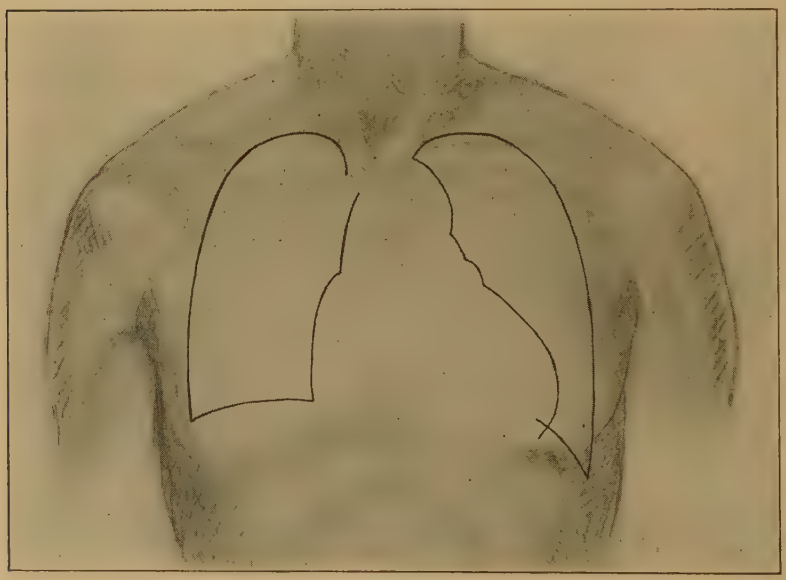


FIG. 260.—Aortitis of the first portion and arch of the aorta, and extreme left ventricular hypertrophy. From a man of 50 with cardiosclerosis and decompensation. The first clinical symptoms occurred six months after a very severe streptococcus pneumonia.

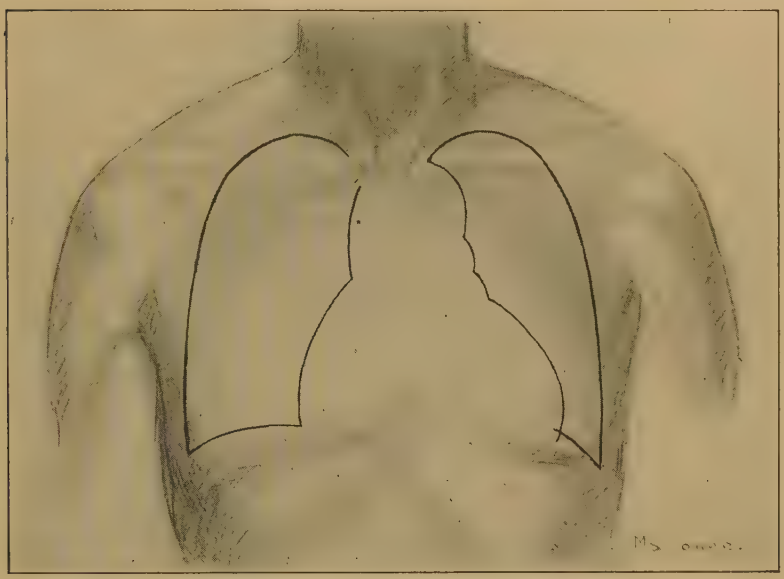


FIG. 261.—Luetic aortitis affecting the arch. Left ventricular hypertrophy. From a patient of 60 with a luetic history and a positive Wassermann reaction.

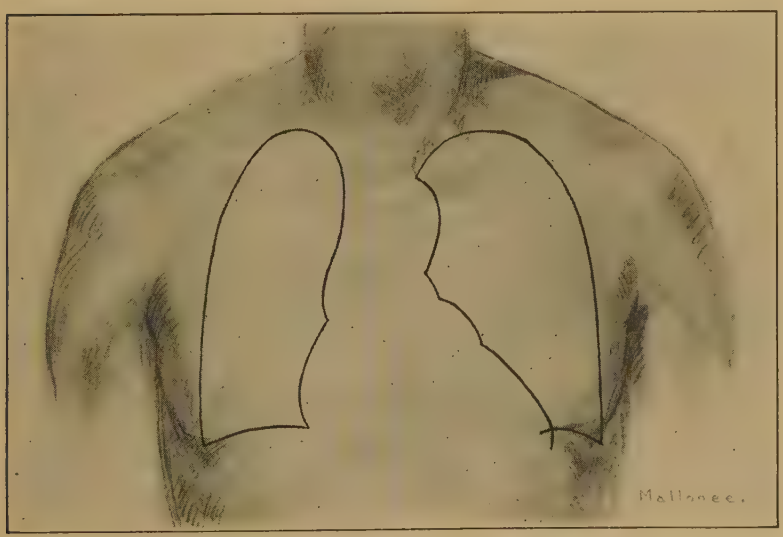


FIG. 262.—Enlargement and low implantation of the first portion of the aorta; moderate enlargement of the left ventricle. From a male patient 58 with chronic nephritis and myocarditis.

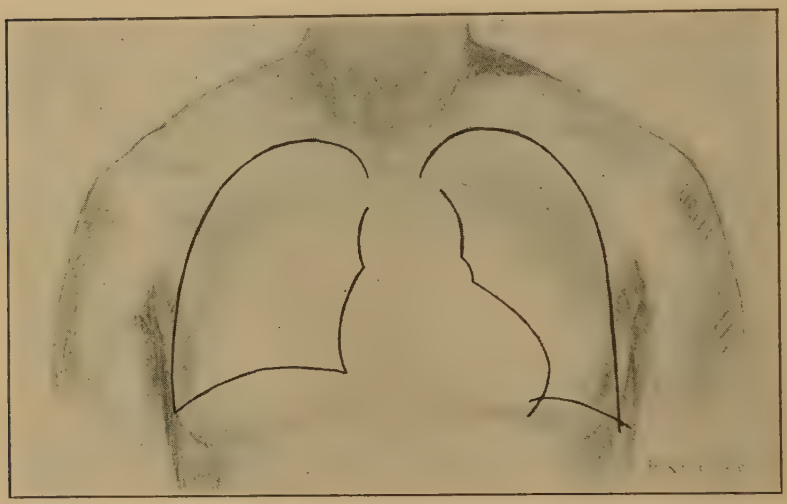


FIG. 263.—Luetic aortitis affecting the arch; left ventricular hypertrophy.

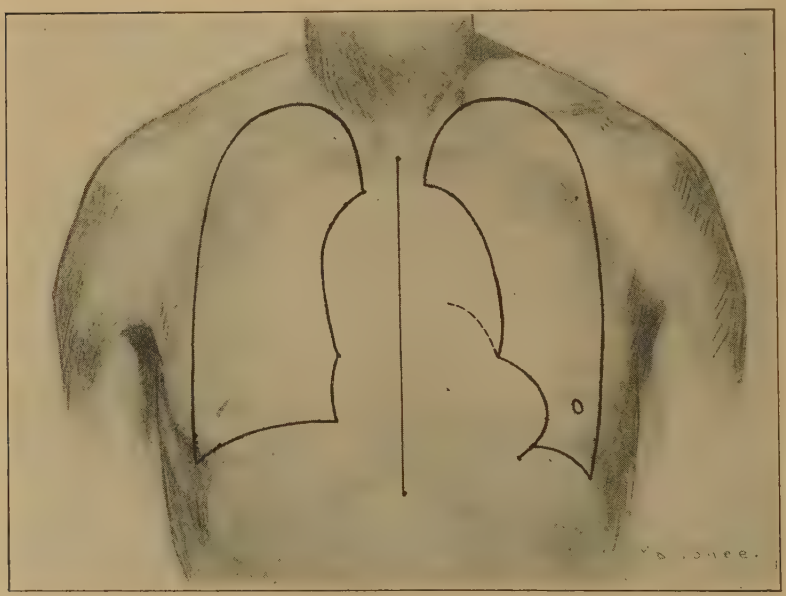
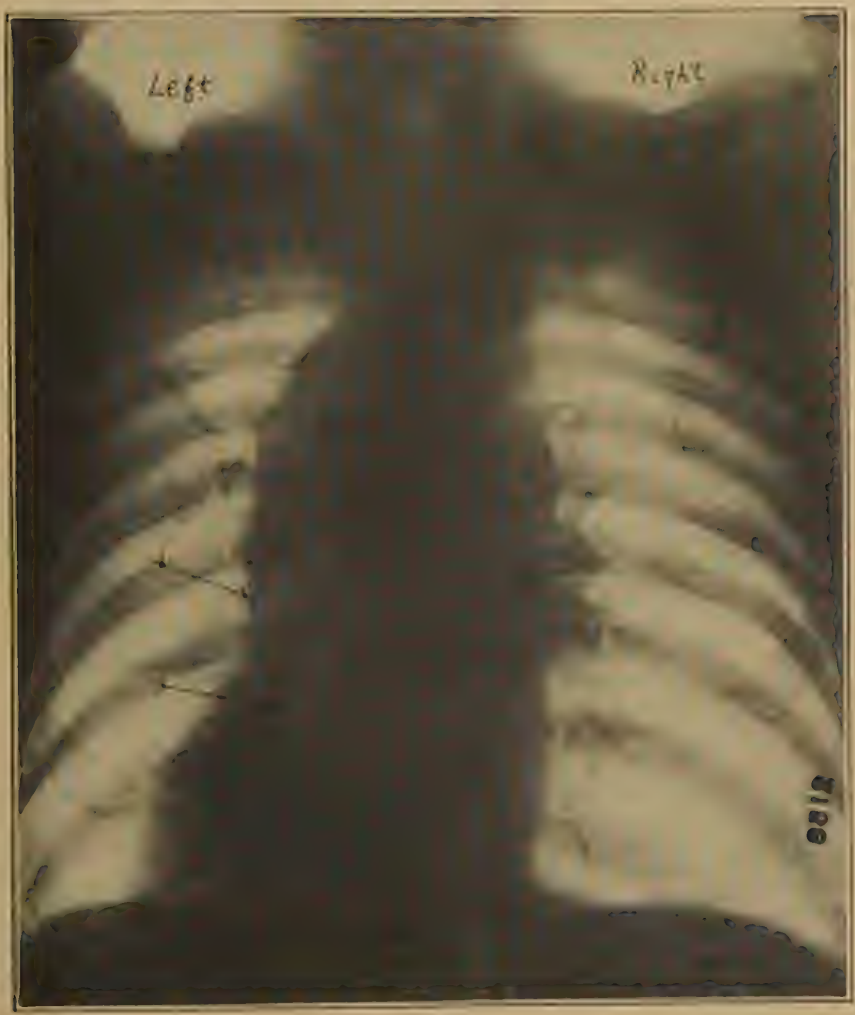


FIG. 264.—Diffuse aneurismal dilatation of the descending thoracic aorta. From a patient of 69 with a history of a luetic infection 40 years ago, and a positive Wassermann reaction of the blood.

Moderate dilatation and enlargement of the first or ascending part of the aorta are characterized by an abnormally broad shadow which originates below the normal level. The left border of the arch is found beyond its usual limits and, in addition, may be sharply curved. The entire aortic




G. 265.—Photograph from X-ray plate, showing aneurismal dilatation of the descending thoracic aorta.

shadow thus becomes abnormally broad. This type of enlargement accompanies aortitis from any cause—senile arteriosclerosis, chronic nephritis with hypertension, luetic aortitis. It is rarely found in rheumatic aortic valvular disease.

As with standards of measurements for the cardiac orthodiagram, similarly there are several objections to a standard measurement for the normal

aorta. Some of these objections are the indefinite point of origin of the aorta, the difficulty at times of deliminating its borders, the high or low position of the arch, the normal variation in size and shape of the heart of each individual. In aortic dilatation of moderate degree, the diagnosis may

FIG. 266.—Photograph of Roentgenogram of aneurismal dilatation of the descending thoracic aorta; left ventricular hypertrophy. From a woman of 50 with a positive Wassermann reaction of the blood.

therefore depend upon abnormal contour rather than upon abnormal size of the vessel. In moderate dilatation of the ascending aorta there is a slight outward ballooning of the right aortic outline; in moderate dilatation of the arch, the curve is broadened, while in that of the descending part of the arch, there is distinct widening of the entire aorta. Finally, there are dilatations

confined to the descending thoracic aorta (Chapter XVI); the latter then assumes a spindle-like or diffuse enlargement, which lies mainly behind the ventricles. The various types of aortic enlargement are best seen and recognized by turning the patient in different directions in order to obtain lateral and diagonal views of the aorta. This is of particular value in differenti-

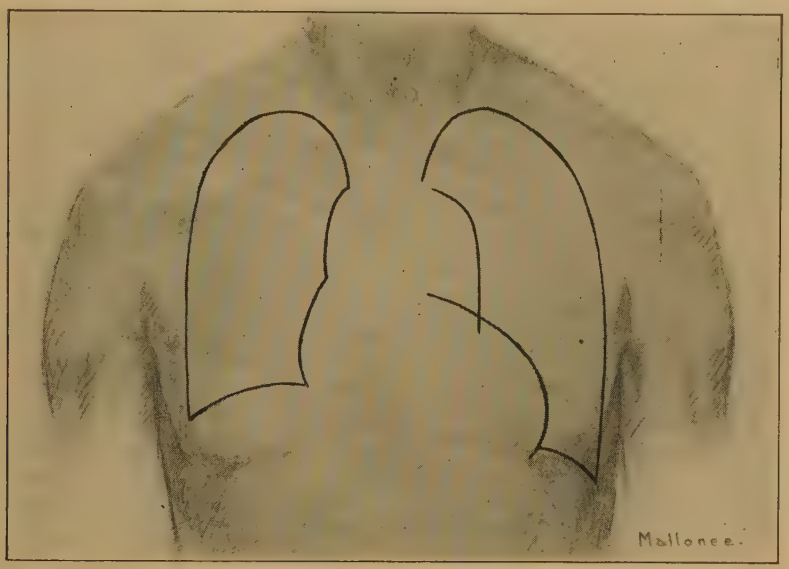


FIG. 267.—Aneurismal dilatation of the arch and descending thoracic aorta.

ating aortic shadows from other structures, for example, from tumors, which in a sagittal direction may resemble and be mistaken for the aorta in area and position.

Orthodiagrams of various types and degrees of aortitis with accompanying cardiac hypertrophy, and of aneurismal dilatations are shown in Figs. 257–267, with a brief summary of their etiology and clinical diagnosis.

The X-ray and orthodiascopic examination of aneurisms is described in Chapter XVI.

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TABLE OF AVERAGES[1]

| No. of cases | Weight, kg.[2] | Height, c.m. | Age, yrs. | Transverse diameter, c.m. | Long diameter, c.m. | Area, sq. c.m. | Angle,[3] deg. |
|---|---|---|---|---|---|---|---|
| 2 | 53 | 154 | 25 | 13.0 | 13.5 | 98 | 37 |
| 2 | 54 | 163 | 30 | 11.9 | 13.2 | 97 | 43 |
| 1 | 55 | 158 | 29 | 12.8 | 14.5 | 122 | 42 |
| 4 | 56 | 167 | 24 | 12.8 | 14.2 | 106 | 44 |
| 4 | 57 | 166 | 23 | 11.9 | 14.2 | 107 | 50 |
| 3 | 58 | 167 | 24 | 11.8 | 13.3 | 100 | 46 |
| 4 | 59 | 169 | 22 | 12.0 | 13.7 | 111 | 46 |
| 9 | 60 | 171 | 24 | 12.3 | 13.8 | 103 | 41 |
| 5 | 61 | 168 | 26 | 11.9 | 13.2 | 101 | 41 |
| 6 | 62 | 169 | 27 | 12.5 | 14.1 | 110 | 44 |
| 11 | 63 | 169 | 28 | 12.6 | 13.8 | 105 | 43 |
| 10 | 64 | 170 | 26 | 12.7 | 13.9 | 106 | 40 |
| 9 | 65 | 171 | 23 | 12.6 | 13.9 | 112 | 43 |
| 6 | 66 | 170 | 23 | 12.4 | 14.4 | 111 | 45 |
| 10 | 67 | 170 | 25 | 13.7 | 14.4 | 110 | 35 |
| 10 | 68 | 173 | 25 | 13.2 | 14.5 | 122 | 41 |
| 12 | 69 | 175 | 28 | 13.3 | 14.5 | 117 | 41 |
| 10 | 70 | 175 | 27 | 13.1 | 14.2 | 108 | 41 |
| 8 | 71 | 172 | 27 | 13.4 | 14.8 | 112 | 38 |
| 9 | 72 | 173 | 23 | 13.2 | 14.5 | 118 | 43 |
| 2 | 73 | 175 | 23 | 14.3 | 14.8 | 114 | 36 |
| 7 | 74 | 174 | 24 | 13.4 | 14.4 | 110 | 38 |
| 8 | 75 | 172 | 25 | 13.2 | 14.7 | 107 | 39 |
| 1 | 76 | 176 | 25 | 14.5 | 15.3 | 121 | 33 |
| 2 | 77 | 180 | 29 | 12.5 | 15.4 | 126 | 48 |
| 2 | 78 | 171 | 26 | 14.2 | 14.5 | 108 | 31 |
| 3 | 83 | 180 | 24 | 13.5 | 15.7 | 123 | 43 |
| 1 | 86 | 180 | 26 | 13.8 | 14.7 | 110 | 36 |

[1] From A. E. Cohn, Archives of Internal Medicine, 1920, XXV, 504. Table of averages derived from the data and measurements of the normal hearts of 161 soldiers.

[2] 1 kg. = 2.2 lbs.

[3] Angle degrees = Angle formed by long axis (L.O.) with transverse axis (M.L.). See Fig. 236A and B.

TABLE SHOWING THE AVERAGE TRANSVERSE DIAMETER CORRESPONDING TO A GIVEN BODY WEIGHT AS REPORTED BY VARIOUS OBSERVERS COMPARED TO THE STANDARD GIVEN IN TABLES A AND B[1]

| Weight | Average transverse diameter | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Standard | Bardeen, sitting | | Veith | | | | Dietlen supine | Otten, supine | Claytor and Merrill, vertical | |
| | | | | Supine | Sitting | | | | | | |
| | | | | | Male | | Female | | | | |
| | | Male | Female | Male | 1 | 2 | | | | Male | Female |
| Kilos | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. | c.m. |
| 15–19 | 8.3[2] | 8.9 | 8.9 | 8.75 | 8.1 | 8.7 | 8.1 | | | | |
| 20–24 | 9.0 | 10.2 | 8.6 | 9.42 | 9.1 | 9.3 | 8.6 | | | | |
| 25–29 | 9.6 | 9.4 | .... | 10.0 | 9.2 | 9.4 | 9.4 | | | | |
| 30–34 | 10.2 | 10.8 | .... | 10.5 | 10.3 | 9.5 | 9.8 | | | | |
| 35–39 | 10.7 | 10.2 | 10.4 | 10.57 | 9.8 | 10.6 | 9.8 | | | | |
| 40–44 | 11.1 | .... | 10.7 | .... | .... | .... | ... | 11.3 | 11.1 | .... | 10.2 |
| 45–49 | 11.5 | 10.8 | 11.3 | .... | .... | .... | ... | 11.4 | 11.4 | .... | 10.2 |
| 50–54 | 11.9 | 12.0 | 11.7 | ..... | .... | .... | ... | 12.4 | 11.6 | .... | 10.7 |
| 55–59 | 12.3 | 12.4 | 12.2 | ..... | .... | .... | ... | 12.9 | 12.3 | 10.9 | 11.0 |
| 60–64 | 12.6 | 12.7 | 12.2 | ..... | .... | .... | ... | 13.1 | 12.7 | 11.8 | 11.2 |
| 65–69 | 13.0 | 13.0 | 14.2 | ..... | .... | .... | ... | 13.2 | 12.7 | 11.8 | 11.1 |
| 70–74 | 13.3 | 12.9 | 12.8 | ..... | .... | .... | ... | 13.4 | 13.0 | 12.3 | 11.6 |
| 75–79 | 13.6 | 13.5 | .... | ..... | .... | .... | ... | 14.3 | 13.2 | 12.4 | 11.9 |
| 80–84 | 13.9 | 13.7 | .... | ..... | .... | .... | ... | 14.4 | .... | 12.9 | |

[1] From C. R. Bardeen, American Journal of Anatomy 1918, XXIII, 448.

Tables A and B contain Bardeen's standard measurements derived from many individuals with normal hearts.

## CHAPTER XIII

## HISTORY RECORD—NORMAL HEART SOUNDS—PHYSICAL EXAMINATION OF THE HEART—ABNORMAL SOUNDS AND MURMURS

**History Record.**—I know of no more important preliminary in the physical examination of the heart than a painstaking history, carefully obtained, of the patient's illness and complaints. This phase of cardiovascular examination is often neglected, yet it is here that the clinician of broad general experience holds a well-merited position, for by his experience he is enabled to evaluate perhaps a prolix story, separating the important from the non-important. Where indicated he may concentrate his attention upon apparently trivial symptoms. He may, for example, be more interested in the gastric symptomatology of a patient with precordial pains and stomach complaints than in the cardiac complaints *per se*, especially if the examiner gain the impression that the cause of the pains lies outside of the heart. The questions then must bring out such complaints and symptoms as eructations, pyrosis, discomfort after meals, local tenderness, etc., particularly if the patient has given no definite history of a rheumatic, tonsillar or scarlatinal infection, the usual forerunners of cardiac disease. Often the history must be pieced together or again gone over after the physical examination has been completed. For instance the latter may demonstrate entire absence of organic cardiovascular disease and yet extrasystoles may be present. More searching questioning may then reveal sudden fright, for example—perhaps long since forgotten by the patient—as the underlying cause of the cardiac irregularity. As has been previously pointed out (Chapter XI), the clinician must possess full knowledge of all the peculiar and varied subjective sensations that extrasystoles produce in some individuals, for these cardiac irregularities may never show themselves even during prolonged or repeated examinations and yet the patient's sensations are so characteristic that, if properly interpreted by the clinician, the presence of the arrhythmia can be correctly diagnosed in the majority of instances. In this connection, I may state that it is very important to know the patient's temperament, especially whether phlegmatic or excitable. One is sometimes deceived into thinking that an apparently phlegmatic manner reflects the individual's temperament. But by further questioning he may elicit the fact that the patient is often aroused and "fussed up" over apparent trifles; or that business worries cause restless nights; or that he is too busy to eat calmly and hence bolts his food; or in the case of women, that apparently trivial household

derangements cause nerve tension and even extreme excitability. I could exemplify these statements by many examples; but common sense, experience and the tact of the examiner alone must guide his questions along the proper channels. I simply wish to emphasize the importance of personal knowledge of each individual's temperament. Temperament is often an extremely important factor not only in the initiation of symptoms but also in their continuance, and especially in their reaction to therapy. This statement applies to organic as well as to functional cardiac disease.

I have not been able to follow any invariable method of procedure in procuring a history record. As an opening question, the usual and most natural one is to discover why the patient seeks medical advice. Since in cardiac cases, the chief complaints are dyspnoea and abnormal chest sensations, one inquires searchingly regarding these: Their duration, mode of onset, peculiarities, what aggravates them, etc. One should especially inquire into the effect of exercise upon dyspnoea and precordial sensations. It must of course be remembered that there are various grades of dyspnoea (Chapter XIV)—slight breathlessness, actual dyspnoea, polypnoea—and that a certain amount of breathlessness accompanies any severe exertion. It is for the clinician to judge how far these sensations are abnormal by observing the patient during rest, both sitting and lying down, as well as the patient's reaction to such exercise as the physician sees fit to employ in order to test the patient's functional capacity (Chapter XIV). One inquires how far the patient can walk comfortably. Do the pains, if present, radiate? Does he get short of breath with their onset? What are the characteristics of the pains: Tearing, agonizing, burning, tingling, etc. (Chapters XXIII, XXIV). Does excitement produce them? Does his heart "thump" or "palpitate" when exercising? Does he avoid stair climbing? These are examples of some suggestive preliminary questions. The clinician of course does not neglect careful inquiry into a history of actual rheumatism, tonsillitis, scarlet fever, syphilis and of alcoholism. I emphasize actual rheumatism, especially inflammatory rheumatism, for there is scarcely any individual who at one time or another, has not had muscular pains. Regarding syphilis, tact is often required, at least in private practice, in order to elicit this etiological factor. With married men, unless the information is volunteered, I inquire regarding venereal disease prior to marriage. With married women, we must usually approach this subject by inquiring about miscarriages, eruptions, sore throats, etc., the usual manifestations of syphilis.

After the history has been obtained, my usual routine is to have the patient rest comfortably in a chair with the chest entirely bared. First I examine the teeth and tonsils. Then I search for enlarged glands about the jaw and palpate the thyroid. The next procedure is the taking of the blood pressure. If hypertension be present and if I believe from the history or from actual observation that the reading has been abnormally heightened because of a nervous element, I either take another reading at the end of the

examination or better still, I allow the cuff to remain on the arm for a few minutes in order to nullify or decrease an abnormally heightened vasomotor tone (Chapter XXV). Next follows the routine physical examination of the heart itself, described later. The lungs are then examined while the patient is still seated. Unless the patient is obviously severely decompensated, the heart is again examined after such exercises as rapid walking, bending, hopping, etc. The type of exercise is guided by the special observation the examiner wishes to make, as for example, the effect upon rate and cardiac activity, upon blood pressure, upon the first appearance of dyspnoea, etc.

The next procedure is to examine the heart with the patient lying comfortably upon the examining table. A routine abdominal examination follows. Points of tenderness, an enlarged liver, gastric distention, visceroptosis are looked for. The physical examination of the abdomen should be particularly searching when gastric symptoms and functional arrhythmias are present. The knee and pupillary reflexes are examined while the patient is still in the recumbent position. Then follows orthodiascopy of the heart, entire chest, lungs and mediastinum. The taking of an electrocardiogram is the next step. While the left leg electrode is still in place the leg is examined for edema. The purpose of pressing upon the tibia should not be made too obvious to the patient. In order to distract attention from the real purpose, I usually inquire whether the pressure is painful. Urinary examination—preferably of a 24 hr. specimen—completes the routine examination. When indicated, but often as a routine, a Wassermann blood examination, and a red and white blood cell examination are made. A chemical examination of the blood, a phthalin test (Chapter XXI) and an examination of the eye grounds may be required in individual instances.

**Normal Heart Sounds.**—The time relationship of the heart sounds in the cardiac cycle has been definitely fixed by the aid of phonocardiograms (Chapter XXXI). The cause of the normal heart sounds is still a matter of dispute, the main question at issue being whether the normal heart in contracting can produce a sound. Weighing physiological, dynamic and phonocardiographic considerations, the most probable cause of the normal first sound is as follows: With beginning ventricular contraction, there is simultaneous contraction of the papillary muscles. At the same time, with beginning rise of intraventricular pressure, the mitral and tricuspid valves close synchronously and noiselessly. This closure is immediately followed by that very brief interval known as the isometric period (Chapter VI) during which all valves are closed. The increasing intraventricular pressure before actual systole (the ejection period), the vibrating valves, the ventricular muscular contraction, are all factors that produce the first part of the first sound; this part of the first sound is transmitted throughout the heart and great vessels. With ejection of the blood (immediately after the isometric period) during ventricular systole, a large fluid column is thrown against the

walls of the pulmonary artery and aorta; this ends and produces the second part of the first sound, at the same time that it changes its acoustic character (Wiggers).

The second sound is usually regarded as being entirely valvular in character, and as due to closure of the semilunar valves. According to Wiggers, these valves close noiselessly and the second sound is produced by after-vibrations of the closed valves caused by the arterial columns in the aorta and pulmonary artery. Some clinical observations, however, do not entirely conform to this theory of the origin of the second sound, at least as far as pathological hearts are concerned. For example, in some cases of mitral disease one may not only hear but actually feel the snappy click of a sharply closed pulmonic valve synchronous with the second sound.

**Third Heart Sound.**—In some young normal adults, a faint third heart sound at the apex in mid-diastole may be heard by placing the patient in the left lateral position. It is soft and low-pitched. Phonocardiograms (Chapter XXXI) of the sound have been obtained. The explanation given for the third heart sound is that it is due to the sudden floating up and tension of the auriculoventricular valves from the first onrush of blood from auricle to ventricle. Other observers (Lewis, Einthoven) have questioned this etiology, so that, for the present, the cause of the third heart sound cannot be considered as definitely established.

The character of the first heart sound in individuals with normal hearts is often regarded as of considerable importance, since upon it wide-reaching conclusions regarding the circulation are based. Thus it is often spoken of as being of "poor muscular quality," or as being "weak." Such conclusions, however are often erroneous, for there are many extraneous physical factors, such as thickness of the thorax, distance of the heart from the chest wall, an intervening pad of lung tissue, configuration of the chest wall, which can "weaken" an otherwise normal and strong first sound. In addition, some so-called "weak" hearts are often surprisingly strong in their contractions (Chapters XII, XVIII). On the other hand if a heart that is obviously hypertrophied causes weak heart sounds, especially in the presence of hypertension, I consider it suggestive of weakened cardiac contractility, for such a heart, when compensating, ordinarily produces a vigorous first sound at the apex.

The normal pulmonic second sound is ordinarily less loud than the second aortic ($P_2 < A_2$). When the reverse is true ($P_2 > A_2$) it is usually regarded as an evidence of an overforceful right ventricle due to disturbance in the pulmonary circuit. This is sometimes true, especially in mitral disease. But, as with the first sound, many physical extra-cardiac conditions may invalidate conclusions based upon an accentuated second pulmonic sound alone, if other important clinical and circulatory factors are not considered. These will be discussed more in detail in conjunction with the various valvular diseases.

**Physical Examination of the Heart.**—Each of the usual methods of clinical examination—inspection, palpation, percussion and auscultation—yields valuable information in appropriate cases. Ordinarily, most emphasis is placed upon auscultation and percussion,—less upon inspection and palpation. In my opinion the two latter, although frequently neglected as routine methods of examination, are often of diagnostic and prognostic importance. Least valuable and most deceptive of all methods is information gained by percussion.

I reserve for later consideration, (Chapter XV) the special abnormal physical signs found in various types of heart disease. Here I shall describe the general information to be gained by following routine procedures applicable to all kinds of cardio-vascular disease.

**Inspection.**—Inspection of the chest and neck frequently reveals important data concerning the cardiovascular system.

**Jugular Pulsation.**—The manner of filling and the degree of distention of the jugular veins is best observed by placing the patient in the supine position. Even pulsation of the thyroid veins may thus become visible. In patients with long-continued decompensation it is common to find the jugular veins tortuous, thickened, engorged and greatly distended; the situation of the venous valves is then well outlined and may be recognized as small knob-like prominences. These evidences of chronic venous stasis are especially marked in old stenotic lesions with auricular fibrillation, or in old patients with decompensated cardiosclerosis. Tricuspid regurgitation from relative insufficiency or valvular disease is sometimes accompanied by engorged or hyperactive pulsatile jugulars, although, as previously mentioned, (Chapter VII), the interposition of the right auricle probably prevents such jugular hyperaction in many instances. In cases of *auricular extrasystoles*, the premature jugular pulsation ($a'$ wave) may occasionally be visible. More often the coincidence of auricular and carotid waves (composite $a'+c'$ waves, Chapter X) found in *ventricular extrasystoles*, is recognized in the jugulars as abnormally large and premature pulsations. A similar jugular wave, not premature in the time of its occurrence, is present in complete heart block when auricles and ventricles beat synchronously ($a+c$ waves; Chapter X). This observation is of use in the differentiation, by inspection, between complete and incomplete heart block, for in the latter there are no such superimposed waves. In auricular flutter the jugular pulsations are usually too faint and indistinct to be counted. However in one case which I observed the jugular veins literally throbbed, and were so engorged that the number of pulsations could be clearly discerned at a distance of several feet. In auricular fibrillation, in addition to the distention already referred to, the jugulars beat irregularly in consonance with the arrhythmic $c-v$ waves.

**The Carotid Pulse.**—The inspection of the carotid pulse is of great importance. By its overaction, pulsatile distention and vigor, it is often

indicative of aortic disease or hypertension; if, in addition, the carotids visibly and markedly collapse with diastole, they indicate an aortic regurgitant lesion. Vigorously throbbing carotids also accompany rapid and violent heart action from any cause, (for example, exopthalmic goiter). But marked carotid hyperaction with a normal pulse rate in the middle-aged or in the old is usually evidence of aortitis or of hypertension; its absence, however, does not necessarily exclude these conditions. This type of carotid action is also seen, although rarely, in young individuals with normal hearts; in them, it often corresponds to strong systolic ventricular contractions which can be very readily observed by fluoroscopic examination. Extrasystoles corresponding to so-called "missed beats" at the radial are sometimes visible as momentary breaks in the otherwise rhythmical carotid pulsations. Occasionally the extrasystolic beat itself is seen as a fainter beat. Grossly irregular carotid pulsations are often seen in auricular fibrillation; these naturally correspond to the usual tumultuous irregular cardiac activity typical of this arrythmia.

**Aortic Pulsation.**—Allied to carotid overaction in clinical significance is aortic pulsation, recognized by the pulsatile rise of the tissues in the jugulum. This indicates more advanced aortic change than does carotid overaction alone. Aortic expansion is most noticeable in aortic regurgitation with left ventricular hypertrophy, and in aneurism and aneurismal dilatation of various parts of the aorta (Chapter XVI), especially during the stage of decompensation. On the chest wall, capillary pulsation characteristic of aortic regurgitation may occasionally be seen. A band of dilated venules and capillaries over the lower anterior part of the chest sometimes accompanies aortic lesions, particularly aneurisms or aneurismal dilatations. Unless well marked, however, they are not characteristic of aortic disease, for they are sometimes found in obese individuals (especially in men) without aortic disease. Large aneurisms that touch, adhere to, or erode the chest wall naturally give rise to visible pulsatile expansion; their usual site is the second and third interspaces and the interclavicular notch, more rarely the left upper interspaces. Aneurismal dilatations of the aorta very infrequently cause erosion, and are rarely adherent. If they touch or approach the chest wall, pulsatile expansion, not noticeable otherwise, may be seen by placing the eye on a level with the chest. By this method, not only dilatations of the first part and arch, but even aneurismal dilatation of the descending thoracic aorta (Chapter XVI) may be discerned as distinct systolic heaves above and partly behind the rise of the left ventricle.

**Inspection of the Apical and Precordial Region.**—Regarding the apical and precordial region, important information can be gleaned by inspection alone. In thin-chested individuals with decompensated mitral stenosis, it is often possible to discern an overacting pulmonary artery which, upon auscultation, produces an accentuated, clicking second sound. Many varieties of abnormal ventricular action are also readily recognized by inspection. Thus

ventricular activity is visible over almost the entire precordium in massive left ventricular hypertrophy, in the violent action of tachycardia, in the ventricular dilatation of the acute stages of valvular endocarditis, or in some congenital lesions. An overactive ventricular impulse may be confined to a small area at the apex in mitral lesions, especially in young individuals. One often finds heaving, broad ventricular impulses in old, decompensated mitral lesions. Obviously the thickness of the chest wall, the posture of the patient and the illumination are considerations which considerably mask or influence these physical signs. In thin-chested individuals or in women with the left breast well raised, it is sometimes possible to distinguish a double systolic impact corresponding to the reduplicated first sound (q. v.) heard on auscultation. Some types of arrhythmias are also recognized by inspecting the apex. The regular, slow ventricular activity of heart block, the irregular ventricular action of auricular fibrillation, the apparent intermission of cardiac activity denoting premature contraction—all these are often distinguishable upon inspection of the apical region. The extrasystolic contraction itself is rarely seen because it is usually too weak to cause a visible ventricular impact. The movement of the apical region during the respiratory phases may likewise be observed in those thin or moderately thick-chested individuals who possess a fair amount of diaphragmatic excursion.

**Inspection of the Right Lower Interspaces.**—Over the right lower chest, aside from aneurisms and aneurismal dilatations already mentioned, very little information can be gleaned by inspection. Right auricular and ventricular enlargement rarely give rise to visible pulsation. I have met with isolated exceptions, however. One case was that of a boy of 17 with a double mitral lesion and a somewhat enlarged liver. At the time of hospital admission he was suffering from an acute rheumatic exacerbation of his endocarditis. To the right of the sternum, in the third and fourth interspaces, there was a visible pulsatile area approximately the size of a pigeon's egg. Over this, there was heard a very loud harsh murmur; a rough palpable thrill was also present; both were systolic in time. Fluoroscopic examination showed that the pulsation was due to a greatly enlarged right auricle. The patient died a few weeks later. At necropsy, a double mitral lesion, hypertrophied right and left ventricles, and a hugely dilated, engorged and thickened right auricle were found. *In situ* in the chest cavity, the longest measurement of the auricle was 17 c.m., its broadest, 8 c.m., (Fig. 268). In another instance of decompensated double mitral lesion in a woman of 26, intensely dyspnoeic, cyanotic, and with a pulsating liver there was a similar but less marked visable auricular pulsation in the third and fourth interspaces; a thrill was also heard over the same area.

Visible epigastric pulsation in patients with dilated and hypertrophied hearts is fairly common. It is usually attributed to hypertrophic enlargement of the right ventricle, but necropsy reports have not always substantiated this supposition.

Liver pulsation as the result of engorgement of the portal circulation is not infrequent in decompensated mitral lesions, or in relative (functional) tricuspid regurgitation. It is less common in aortic lesions. The pulsations may be sufficiently gross to be seen at a distance; at other times close examination, with the observer's eye placed on the level with the patient's abdomen, is necessary to discern the pulsating liver. Auricular fibrillation may occasionally be diagnosed by inspection of the liver, if its pulsations are mani-

FIG. 268.—Heart with hugely dilated right auricle (see text). A, dilated right auricle; B, midline of body; C, enlarged right ventricle.

festly and grossly arrhythmic. In one instance of heart block, I was able to see the venous auricular pulsations as distinct, small rhythmic pulsatile rises. That they were actually auricular pulsations was proven by taking polygraphic tracings with a large "venous" cup over the liver (a liver cup), and by electrocardiograms showing heart block.

**Palpation.**—Palpation in the neck often substantiates the information gained by inspection. Extrasystoles may be felt in the carotids when they are missed at the wrist. Overacting hearts (for example, in exopthalmic goiter) often produce palpable carotid thrills; these may be due to whorls and eddies from the violence with which the blood is thrown into the arterial circulation. Such short, sharp thrills must be distinguished from the steadier, longer continued ones accompanying rheumatic aortic stenosis, and from arteriosclerotic roughening and thickening of the aortic valves and walls. The typical quick filling and collapsing pulse of aortic regurgitation can also be readily diagnosed by palpation of the carotids alone. A point insufficiently

emphasized is the knowledge regarding arteriosclerosis which may be gained by carotid palpation; the condition of the external carotids whether tortuous, thick, hard, and non-elastic, or the reverse, is thus often determined. Similar information is occasionally derived by palpation of the common carotid.

**Aortic Pulsations.**—With a normal cardio-vascular system and an aortic arch situated moderately high in the chest, the finger tip placed in the interclavicular notch and insinuated behind the manubrium recognizes aortic pulsation as a soft quiet impact. In marked hypertension a sense of sharp fling is often given to the examining finger. In luetic aortitis with aneurismal dilatation, there is a feeling of a broad, strong aortic impact, especially when decompensation is present; this is sometimes combined with a palpable thrill. The rough thrill accompanying aortic stenosis, and the sharp rise and subsequent collapse of the aortic walls in aortic regurgitation, can also be diagnosed by palpation in the jugulum.

Palpation over the right base of the heart may be of great diagnostic importance. In extreme cases of rheumatic aortic stenosis, a rough rasping systolic thrill is readily felt in the second and third right interspaces. The overacting aorta in aneurism and aneurismal dilatation of the first part of the aorta (Chapter XVI), and the sharp aortic pulsatile distension in hypertension, are sometimes palpable over the same area. If present in the former, it probably denotes an extremely roughened, thickened aortic wall, or extreme dilatation. Bulging of the right anterior chest, found in adherent or erosive aneurisms, is usually palpable. Pulsations of aneurisms or aneurismal dilatations of the descending part of the arch, or even of the descending thoracic aorta, may occasionally be felt by placing the fingers snugly against the chest over the base of the heart on the left side, or in the third and fourth left interspaces. In the latter position, the location of the impact area near the sternum, and the time of its appearance, serve to distinguish it from the impact due to ventricular systole (Chapter XVI).

In congenital pulmonary stenosis and in patent ductus arteriosus, thrills, usually systolic in time, are often palpable over the left base. In congenital patent interventricular septum, there is a rough, harsh thrill, systolic in time, palpable over the entire precordium, but especially over the lower sternum. In patients with dilated and overacting pulmonary arteries, the sharp click-like closure of the valves can occasionally be felt by palpation over their site. This condition, combined with a murmur over the same area, is common in mitral valvulitis in children, in whom it is sometimes incorrectly diagnosed as a congenital pulmonary lesion. Similar hyperaction is occasionally present as a temporary phenomenon in normal individual.

**Palpation in Mitral Lesions.**—In typical mitral stenosis, with the heart beating rhythmically, there is a short presystolic thrill or occasionally a long diastolic one palpable in the apical region. When the auricles fibrillate and the ventricles beat irregularly and tumultuously, an intense rough thrill, most prominent at the apex and occupying a varying part of the diastole,

may be felt; if the irregular ventricular activity be controlled by digitalis, the thrill becomes less marked or it may entirely disappear. A palpable systolic thrill over the lower precordium not infrequently accompanies rheumatic regurgitant lesions, and may indeed be present when the murmur itself is faint or absent. This condition was found, for example, in a young girl without any previous rheumatic history; she had had frequent attacks of dyspoena, and edema of the face and extremities. At the apex a scarcely perceptible murmur was heard; it was not transmitted. Over the lower precordium there was a very pronounced palpable thrill. Systolic precordial thrills may also be felt when the mitral cusps are thickened or covered with lime deposits as the result of a cardiosclerotic non-rheumatic process (Chapter XVII).

Further information regarding the character of ventricular contraction and of the apex beat may be gained by applying the flattened palm snugly over the lower precordial area. The booming broad ventricular shock and diffuse apical impact of hypertrophy may thus be readily diagnosed, particularly if the heart action be normal in rapidity or be abnormally slow. Rapid action, even of a normal sized heart, usually gives the impression of enlargement, probably because of the rather violent impact against the chest wall. I have often corroborated the normal size of such hearts by fluoroscopic examinations. A somewhat thrill-like sensation is imparted to the examining hand during systole by the violent overaction which usually accompanies tachycardia from any cause. Here the thrill may be due to sharp vibratory action of the auriculo-ventricular valves transmitted through a thin chest wall or one of moderate thickness. It is sometimes possible to recognize the weakened impulse of an enlarged, dilated heart by palpation. A double systolic apical impact, corresponding to the reduplicated first sound (so-called gallop rhythm, q. v.), when due to left ventricular hypertrophy, hypertension or to aortic valvular lesions, may also be distinguished by palpation over the apex. The etiology of these reduplications will be discussed in connection with auscultation. *Pulsus paradoxus* (Chapter XI) may be diagnosed by palpation, for there is a gradation in the strength of ventricular contractions corresponding to the rhythmic waning and waxing of the pulse volume with inspiration and expiration, respectively. This is commonly attributed to pericarditis with effusion or to intrathoracic tumors. I have observed it in occasional cases of myocarditis with severe decompensation, or in the late stage of cardiac failure.

Finally a double valvular click, occasionally present and due to asynchronous closure of the individual leaflets of the aortic or pulmonary valves, may sometimes be diagnosed by palpation over the respective sites of these valves. This sign is present oftener over the pulmonary than over the aorta.

**Percussion.**—To the ordinary and older methods of finger and pleximeter hammer percussion have been added the auscultatory, and more recently the flexed finger (Goldscheider's orthopercussion) methods. The auscultatory

method consists in placing the bell of the stethoscope over the center of the sternum and noting when scratch marks or light tappings made by the finger nail or finger tips first become audible. Orthopercussion consists in light perpendicular tapping over a sharply flexed finger held perpendicular to the chest wall, the finger acting as the pleximeter. These two methods have been advanced as refinements by which the exact cardiac border could be more delicately and accurately outlined.

**Inaccuracy of Percussion Methods** (Figs. 269–271).—Careful and continued use and observation of the above special as well as the usual methods of percussion, checked by comparison with the unequivocal standards furnished by orthodiascopy and fluoroscopy, have convinced me that all methods of percussion are inaccurate and unreliable even for clinical purposes. Besides varying among themselves considerably in accuracy, there is no means of judging in advance which method will prove sufficiently exact in any individual case. In many instances, I have requested experienced and excellent clinicians to map out the cardiac border according to their own favorite method of percussion, and I have demonstrated to them by fluoroscopy that the results of their examination were rarely of sufficient exactness even for general bedside purposes. These statements apply to percussion for superficial dullness as well as for flatness. For example, very marked aortic dilatations were at times entirely overlooked; definite extension of the cardiac border to the right was not even approximately delimited. My own gross errors by the various methods of percussion have included practically all miscalculations: The delimination of the size and of the upper border of the aorta, the right border of the heart, and the upper ventricular border. Figures 269–271 demonstrate some of these errors in patients with normal hearts, in whom mistakes of outline varying from several centimeters to almost grotesque proportions have been committed. From the viewpoints of physics and acoustics, there seem to be good reasons for diagnostic errors in the use of percussion. From the physical standpoint, the heart may be succinctly regarded as a blood-containing, more or less soft bag situated at various depths from a chest wall of varying thickness. Between the chest wall and the heart lies a lung pad of varying dimensions. The great vessels —the aorta and the pulmonary artery—are of such heterogenous densities that the sound evoked by percussion of the chest wall can but rarely give reliable data regarding the size and shape of the heart; tissues of different densities do not conduct sound evenly, hence it is impossible to state which of the many acoustic variants actually cause the differential dullness. Unless greatly enlarged and near the surface, the aorta, situated normally, mainly behind the sternum, is scarcely sufficiently thick-walled or, with its contained blood, of sufficient density to cause differential dullness or flatness on percussion. Similar reasons make difficult the delimination by percussion of the right cardiac border, consisting of the right auricle below and the great vessels above (Chapter XII). Percussion of the left ventricular

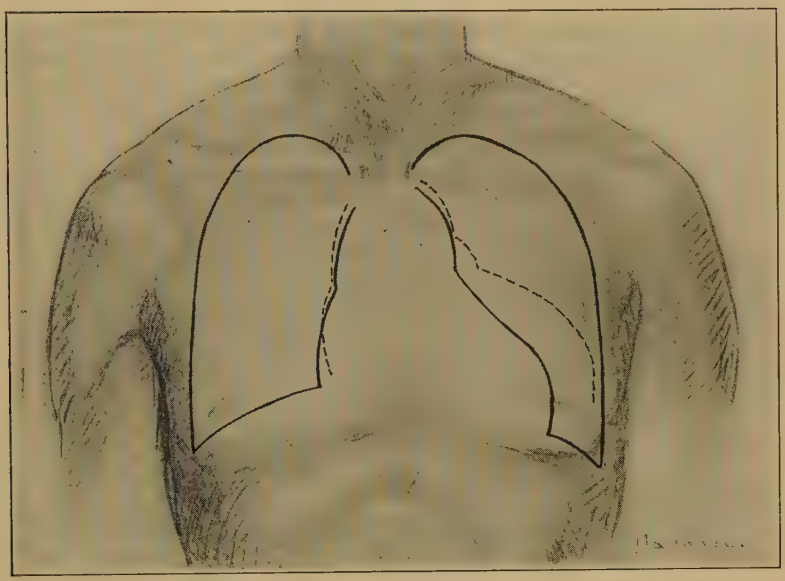

FIG. 269.

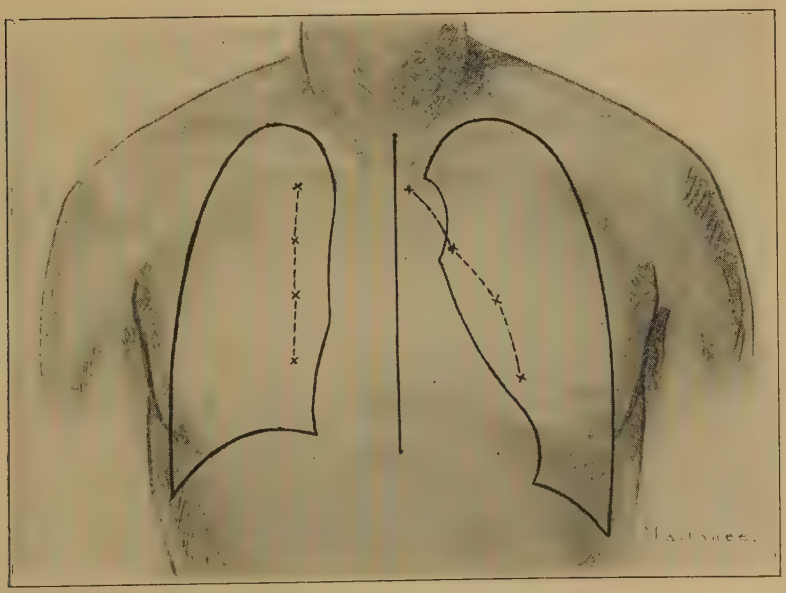

FIG. 270.

area is surrounded by less difficulties, for the ventricular mass is denser and more uniform; but its approximate size and contour—whether squatty, broad, erect or flat, subjects already discussed in connection with orthodiascopy (Chapter XII)—are considerations which I believe are beyond the physical limits of percussion. Frequent comparisons between percussion and radiographic findings have also convinced me that the left border of the heart may actually be several centimeters beyond the percussed outline, and that the lowest point of the heart—the apex as viewed fluoroscopically

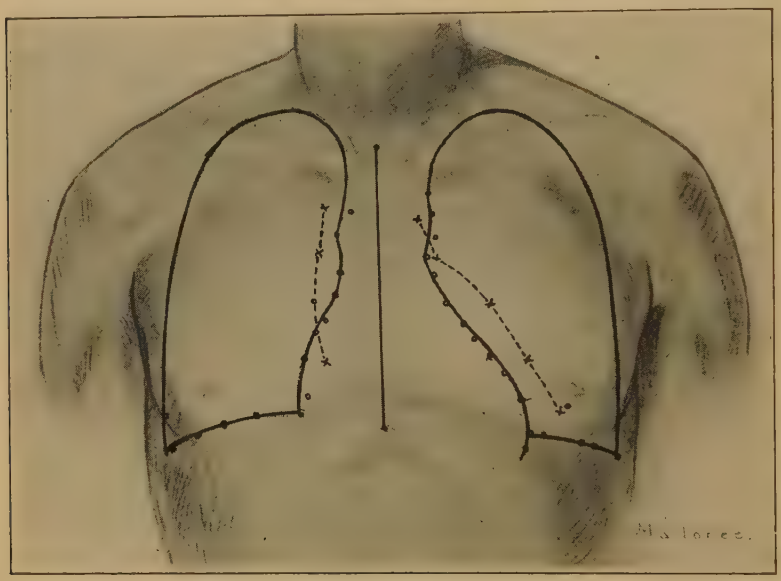

Fig. 271.

Figs. 269, 270 and 271.—Inaccuracy of percussion methods. The dotted outlines show the heart delimited by percussion. The solid lines show the actual cardiac contour obtained by orthodiascopy.

at the end of inspiration—is usually from 2 to 4 c.m. below its position as located by percussion or palpation. This difference between the "palpable" and "fluoroscopic" apex is especially noticeable in tall, lean individuals with flat chests, narrow elongated hearts, and mobile diaphragms.

**A Rational Method for the Determination of the Cardiac Outline.**—In view of the limitations and of the inaccuracy of percussion methods, I have attempted to apply the more exact knowledge gained by roentgenography to bedside study of the cardiac outline. The routine I use for this purpose is as follows: First, inspection, and second palpation of the precordium. Then auscultation to determine the presence and type of a possible cardiac lesion. Finally the mapping out of the cardiac outline.

I begin mapping out either above or at the apex. Assuming that we are starting with the latter, I determine the point of the maximal apical impulse.

In the normal adult heart, if it cannot be readily seen or palpated, it can be fixed by auscultation as the point at which the heart sounds at the apex are loudest. In children, the maximal apical impulse is readily determined by palpation. As determined by fluoroscopy, the apex, regarded as the lowest point of the heart, is situated from 2 to 4 centimeters below the site of maximal impulse. This variable distance depends on the amount of diaphragmatic excursion and on the type of cardiac contour. The former can be roughly estimated by the structure of the chest and the amount of chest expansion. The cardiac contour is apt to correspond to the general physique of the individual. In the thickest, with short extremities and pudgy hands, the ventricular outline is apt to be broad and flat, and to lie with a large part of its lower ventricular surface on the diaphragm; diaphragmatic mobility is usually limited, therefore the lowermost point of the heart and the maximal apical impulse roughly correspond. In the slender and agile with elongated cardiac contours, diaphragmatic mobility is usually marked, the lowermost part of the heart being much below the area of the maximal impulse. If the latter type of individual does not possess an elongated heart, that fact is often disclosed by strong and forceful cardiac action, and the cardiac contour is determined accordingly.

Thus, by gauging the two variants—diaphragmatic mobility and the type of cardiac contour—the position of the actual apex may be determined with a fair degree of accuracy. When thus determined, that point is marked upon the chest wall.

The next fixed line to be marked is the highest part of the aorta corresponding to the upper level of the aortic arch. The tip of the finger is introduced as deeply as possible behind the sternum. If aortic pulsation is plainly palpable the aorta is regarded as "high" and the upper line of the arch is situated within a centimeter below the tip of the palpating finger. If aortic pulsation is not plainly palpable, there is low implantation of that vessel. The point at which aortic sounds are heard most prominently is then sought as a landmark; the upper limit of the aorta is usually about two centimeters above this area. After the upper aortic limit is determined by either of these methods, it is marked across the sternum. When viewed fluoroscopically, it may be roughly estimated that the upper two fifths of the right cardiac contour consists of the right aortic curve, and the lower three fifths of the right auricular curve (Fig. 236). Normally the ascending aorta, seen fluoroscopically, extends about two centimeters from the right sternal border and is visable a distance of about 4 to 6 centimeters—that is, this measures the distance from the upper aortic limit to the point at which it touches the upper border of the right auricle. In thin individuals with long and slender hearts, frequently also in those with broader hearts, the right auricle extends but a slight distance—a centimeter or two—beyond the right border of the sternum and forms a gently convex curve. In short chested, pudgy individuals, the right auricle commonly forms a sharper, more convex curve,

extending as much as 4 or 5 centimeters from the right sternal border. From these considerations, the right cardiac border is marked upon the chest wall.

The arch of the aorta does not usually extend as low on the left as does the aortic curve on the right. In fact, a similar leftsided distance usually includes the curve of the pulmonary, contiguous to the lowermost point of the arch, (Fig. 236). Depending on its contour, the aorta may make a smooth, somewhat long curve, which stretches but a centimeter or two beyond the left sternal margin; or it may be a sharp, knoblike structure, extending 4 or 5 centimeters to the left of the sternum. I have as yet found no method of determining at the bedside the probable contour of this part of the aortic curve.

After the right side of the heart and the aortic arch have been drawn upon the chest wall, the left auricular, and especially the left ventricular curves are blocked in. The former is very variable in extent, fluoroscopically, and in this bedside clinical method, cannot be separated from the left ventricular curve. The latter curve will depend for its contour on the physique of the individual. Here, ordinary percussion may be an added aid in delimiting this part of the left ventricular outline. People who are short and muscular usually have broad hearts, and the ventricular curve is correspondingly flat; those with graceful thoraces and limbs have narrow, more erect hearts and narrower aortæ, hence the left ventricular curve is more elongated.

I have thus far dealt with the contour of the normal heart. I shall only briefly touch upon this method as applied to diseased hearts. In old quiescent mitral regurgitant lesions that have never decompensated, and that present no evidence of hypertrophy, with no booming first sound or heaving impulse, the contour and size of the heart is approximately that of a correspondingly normal heart in a person with the same physique (Figs. 241, 242) for I have frequently found that such mildly diseased hearts are identical with the normal in contour and size. In old decompensated mitral regurgitation, the left ventricular curve is more rounded, the right auricular curve larger, thus the entire heart is broader and more rotund (Figs. 247, 248). In old decompensated double mitral lesions, especially with auricular fibrillation, the left cardiac border is enlarged to the left; less, in a downward direction; the right auricle makes a sharper, rounded curve, so that the entire cardiac outline, including its vessels, assumes an extremely rounded form (Figs. 243–245). This knowledge is important, for the enlarged right auricle can rarely be percussed properly. The general knowledge derived from orthodiascopy regarding the type of contour and size of these hearts, if remembered, will help us considerably in blocking in the cardiac outline after the position of the actual apex and the upper aortic margin are determined in the manner already described.

The typical left ventricular hypertrophies which accompany rheumatic aortic lesions (especially double aortic lesions) usually give us definite visible and palpatory evidence of the enlargement. Booming and reduplicated

FIG. 272.—Solid outline—actual orthodiascopic contour; dotted line—contour mapped out by author's method; functional tachycardia in a man of 40.

FIG. 273.—Solid outline—actual orthodiascopic contour; dotted outline—contour mapped out by author's method; normal heart of a man of 35.

sounds at the apex occasionally give the impression of extreme enlargement, while the fluoroscope will show an aorta only slightly enlarged, but with tremendous systolic excursions. This should be recalled in clinically mapping out the aorta in such cases, and it should also be borne in mind that the palpating finger in the jugulum registers and feels this fling, and hence does not localize the line of the actual upper aortic limit. In other words, such abnormal aortic excursion must be taken into consideration, in determing the actual upper line of the aorta.

Another important fact is that rapidly beating hearts, or those overacting, almost invariably give the impression of enlargement. I believe they

Fig. 274.—Solid outline—actual orthodiascopic contour; dotted line—contour mapped out by author's method; normal heart of a m n of 42.

too, can be mapped out according to the rational scheme already referred to. Otherwise, the impression gained by auscultation alone will almost inevitably lead to erroneous deductions as to the actual size of the heart.

Since following what appears to me this logical and rational method of mapping out the heart, I have been saved from many of the pitfalls and errors into which I had previously fallen. The method follows no rule-of-thumb. It presupposes some knowledge of heart size actually seen fluoroscopically and orthodiascopically, gained from a study of these subjects. The chief objection is that it is not mathematically exact. I should rather call it a scientific gauging and weighing of varients, some of which can, after all, be quickly and fairly accurately determined at the bedside. On the whole I have found the conclusions derived from this method to be much less erroneous, and

therefore much more satisfactory than those derived by practicing various percussion methods. Figs. 272–274 are examples of actual orthodiascopic cardiac contours compared with the outline derived by the method I have just described.

## AUSCULTATION

Despite drawbacks and opportunities for error, auscultation is the most accurate of the four methods for the detection of the type of valvular or myocardial disease. Indeed, the diagnostic aid given by murmurs is often overlooked or minimized, because many individuals with valvular disease and murmurs are compensated and carry on as well as those with organically sound hearts. The presence of cardiac murmurs (by which are meant adventitious sounds, not merely modifications of the normal), their time relationship to events in the cardiac cycle, their intensity, quality, pitch and timbre, and the direction of their propagation anteriorly, laterally and posteriorly as well as through the larger arteries, the presence of audible thrills and rumbles, all require auscultation for their recognition. It is important to remember that murmurs may change in character with change in the patient's position as well as with the state of compensation. They are usually more intense when the patient is lying down; this may be because in other positions (standing or sitting) the apical impulse masks the murmur. Rapid heart action commonly intensifies the murmur of a mitral stenotic lesion, although its duration is considerably diminished. For purposes of careful auscultation, one may attempt to slow the cardiac rate by having the patient breathe deeply and slowly; and to accelerate the heart action, by asking the patient to take a few rapid breaths or to exercise. Another consideration which occasionally modifies both normal sounds and murmurs is the amount of pressure used when applying the stethoscope to the chest wall; hence care must be exercised in applying the bell of the instrument to the chest wall.

**Valvular Murmurs in Rheumatic Endocarditis.**—Organic valvular defects do not always produce murmurs. It is sometimes difficult from auscultation alone to determine which valve is affected, or to distinguish valvular from non-valvular affections. Differential criteria applicable in such instances will be discussed later. As further complicating factors in auscultation the intensity and propagation of the murmurs are considerably modified by the state of cardiac compensation, the amount of dilatation and hypertrophy, the rapidity of heart action, the thickness of the chest wall and the position of the patient.

Organic valvular murmurs are best grouped into those due to narrowing of the caliber of the valvular orifice (stenotic lesions) and those allowing leakage (regurgitant lesions or valvular incompetency).

**Murmurs of Mitral Regurgitation (Mitral Incompetence).**—A mitral regurgitant lesion produces a murmur systolic in time; the second pulmonic

sound is often accentuated. The murmur may be soft and blowing in character, occupying only a part of the systole, and accompanying, not replacing, the first sound. When typical and well marked, the murmur is rough, low or rather high pitched, occupies the entire systole, replaces the first sound, is heard loudest in the apical region and is transmitted laterally to the axilla. More rarely it is heard over the entire precordium. It is sometimes heard posteriorly between the angle of the left scapula and the spinal column; this is especially true in children. The murmur may be accompanied by a palpable thrill over the mitral area. In decompensation with cyanosis, a diastolic murmur transmitted downward (the Graham-Steele) is occasionally heard at the second right interspace; it is probably due to dilatation of the pulmonary orifice with consequent relative insufficiency of the pulmonary valve.

**Murmurs of Mitral Stenosis.**—In this lesion the second pulmonic is frequently accentuated as compared with the second aortic sound. The murmur itself is caused by auricular contraction forcing blood through a more or less narrowed mitral orifice; the name, auriculo-systolic, given to this murmur many years ago by Gairdner, aptly describes its mechanism. It is characteristically a rough, rasping, vibrant murmur. The first sound at the apex is often accentuated to a varying degree. Frequently there is a reduplicated second sound at the apex. The murmur gives the impression of being crescendo in character, but electrocardiographic sound records (Chapter XXXI) tend to show that this "crescendo" quality is only produced by its proximity to the loud first sound.

Some confusion has been brought about by various descriptions of the time relationship of the murmur of mitral stenosis as presystolic, middiastolic, late diastolic and diastolic. In a sense all these are presystolic since they occur at some time before systole. The term presystolic should be applied only to murmurs *immediately* preceding systole, a characterization which clarifies the time relationship of this and the other murmurs. In typical instances, the murmur is heard immediately preceding systole (presystolic) or in late diastole. It is occasionally mid-diastolic, with a distinct gap between its completion and the following systole. This has been demonstrated by electrocardiographic sound records from patients in whom there was prolonged auriculo-ventricular conduction time (usually caused by digitalis); the murmur then occurred at the time of *auricular* systole, which in such instances bears varying time relations to the diastole. A similar instance came under my observation—a young woman with mitral stenosis and gastric symptoms. Electrocardiographic tracings showed progressively prolonged P-R intervals (Chapter X), so that at times there were independent auricular contractions; the rough, rasping murmur was always synchronous with auricular systoles, and, depending upon the conduction time, was heard in varying places in diastole. Such observations clinch the cause of the mitral stenotic murmur as due to auricular contraction. A mitral stenotic

murmur often varies in intensity and duration. These variations probably depend upon the difference in pressure existing in the auricular and ventricular chambers. In the normal heart, at the beginning of ventricular diastole (and therefore of ventricular filling), the auricular pressure is high, the rate of flow more rapid (Chapter VI). With the filling of the ventricle, the difference between ventricular and auricular pressures becomes less and less until the advent of auricular systole which of course precedes ventricular systole. At the same time there is a sharp rise of auricular pressure and an increased rate of flow into the ventricle. Applying these facts to the slow rhythmically beating heart with moderate mitral stenosis, the differential auriculo-ventricular pressure is manifest only at the moment of auricular contraction, consequently the murmur here is presystolic alone. With more marked stenosis or with more rapid heart action (shortened diastole), the differential pressure favors the auricle at the immediate completion of ventricular systole; hence the murmur occurs not only in presystole but also in early diastole, or it may occupy the entire diastolic period. In addition a Graham-Steel murmur (Chapter XXXI) is present in a certain proportion of advanced mitral stenotic cases.

It should be emphasized that the murmur of mitral stenosis can vary considerably; it may even be entirely absent on some days and present on others. Various expedients may then be resorted to in order to bring out or intensify the murmur when mitral stenosis is suspected. A simple procedure is to lay the patient on his left side; this sometimes intensifies the murmur. If the condition of the patient allows it, various types of sharp exercise (*e.g.*, hopping, bending, walking stairs) may be resorted to in order to race the heart and increase its activity for a few minutes. Another expedient that is sometimes tried is to have the patient inhale a pearl of nitrite of amyl. It seems to me, however, that any of the other simpler methods will accomplish the same purpose without the possible disagreeable after-effects of amyl nitrite.

It is especially in auricular fibrillation that the murmurs of mitral stenosis undergo marked changes, for the absence of rhythmic auricular contractions in this type of irregularity precludes the usual presystolic element found in the rhythmically beating heart. With comparatively slow and fairly regular ventricular action, there is a distinct pause between the diastolic murmur and the following systole. Or the murmur may occupy early diastole alone and gradually wane and tail away with the filling of the ventricles, for then intraventricular pressure increases as compared with intra-auricular pressure. A rough, rasping diastolic murmur and a palpable thrill may lose these characteristics under the influence of digitalis, to become much softer and scarcely audible. The reason for this change seems due to steadier ventricular contraction produced by the drug, with consequent lessened differences in auriculo-ventricular pressure. When the ventricle beats very irregularly and rapidly, the murmur is short, sharp and seemingly

occupies the entire shortened diastolic period. With extremely rapid and tumultuous cardiac activity the murmur may become barely audible or distinguishable, although in the occasional longer diastolic pauses its usual characteristics are evident.

**Murmurs of Aortic Stenosis.**—It must be remembered that systolic murmurs over the right base can be due to causes other than actual aortic stenosis. Thus, as already pointed out, functional murmurs in this area not uncommonly accompany anemia or cardiac overaction without valvular disease. The murmur is also present in aortitis accompanying cardiosclerosis (Chapter XVII) and in aortic aneurisms or dilatations, for here the valvular surfaces are roughened and thickened. Typical auscultatory signs over the right base are therefore necessary in order to diagnosticate rheumatic aortic stenosis, especially since this lesion is rare as an isolated rheumatic valvular defect. A radial tracing may show an anacrotic notch (Chapter VII). The impulse at the apex is forcible and gives the palpating hand the impression of gradually reaching its climax.

**Murmurs of Aortic Regurgitation—Aortic Incompetency.**—This murmur varies considerably in intensity and in its area of propagation. When typical, it is diastolic in time, occupies the entire diastole, and replaces the second sound. Occasionally it occupies only part of the diastole. In addition there is sometimes a booming apical impulse. To judge from experiments on animals, the intensity and duration of the murmur seem to be roughly parallel to the degree of regurgitation. The murmur is most often blowing in character; it may be very soft or, occasionally, quite loud and rough. The usual area of greatest intensity is at the right base; however, it is often heard loudest at the second and third interspaces near the sternum. Not infrequently an aortic regurgitant murmur has exactly the characteristics of a mitral stenotic one; that is, it produces a presystolic sharp rumble at the apex (Austin Flint murmur). Under such circumstances, the regurgitant murmur at the base may be faint or entirely absent. Many theories have been advanced for the etiology of the so-called Austin Flint murmur, but as yet no satisfactory explanation has been given. The consensus of opinion is that it is due to narrowing of the mitral orifice from the regurgitant stream of blood in the ventricle. In addition to the diastolic murmur at the base there is often a systolic murmur at the apex in advanced cases of aortic regurgitation. This murmur is probably caused by relative incompetence of the mitral ring and consequent regurgitation during ventricular systole.

**Tricuspid** valvular lesions are very infrequently encountered. The etiology is rarely rheumatic; more often it is of streptococcic, gonococcic or leutic origin. Although both tricuspid regurgitation and stenosis are very rare, the former is the more frequent. The regurgitant lesion is characterized by a blowing systolic murmur, heard best to the left of the lower sternum and transmitted to the right; it is not transmitted posteriorly. It is often accompanied by a pulsating liver and marked jugular pulsation.

I observed one case in which a distinct palpable thrill was also present. Tricuspid stenosis is exceedingly rare and has never been found as an isolated lesion; it has been very infrequently diagnosed during life. The characteristic murmur is described as a presystolic rumble most prominent in midsternum or over the xiphoid. The lesion may be suspected in the presence of extreme cyanosis, right auricular hypertrophy and marked pulsation of the liver and of the veins of the neck.

**Pulmonary valvular insufficiency** is exceedingly rare; it has never been observed as an isolated lesion and has been correctly diagnosed in but a very few instances. The murmur is described as blowing in character, diastolic in rhythm, heard best over the left sound and third interspaces, and transmitted downward. It is usually accompanied by other endocardial murmurs.

**Pulmonary Stenosis** commonly occurs as a congenital lesion (Chapter XIX). Its differentiation from aortic stenosis, which sometimes presents similar auscultatory signs, may be made from the fact that the murmur of the latter is propagated along the carotids, while the pulmonary stenotic murmur is transmitted to the left.

A combination of lesions of the various valves, especially of the aortic and mitral, is very common. The lesions in themselves tend to cause inorganic or so-called functional murmurs (q. v.) from the various degrees of ventricular dilatation they produce. In addition the valvular murmurs may not be typical; therefore it is sometimes difficult or even impossible to determine the exact valvular lesions which are present. The problem is further complicated by the frequent coexistence of pericarditis with its adventitious sounds. An extremely valuable guide in the differentiation of such complicated murmurs is careful comparative auscultation of the differing quality, pitch, and propagation of the murmurs. In this manner, the characteristic murmur of one of the valvular defects may be followed through an apparent maze produced by the presence of other valvular murmurs. Furthermore much information is gained by precordial palpation which, with auscultation, may act as guides in the determination of enlargements typical of the various valvular lesions. Blood pressure observations (Chapter XXV) are also of value in the differentiation. This is especially true of aortic regurgitation in which the difference between the systolic and diastolic pressure of the femoral artery is often much beyond the normal.

**Murmurs in Cardiosclerosis.**—Besides rheumatic valvular affections, atheromatous changes in the mural endocardium, in the mitral valves, and in the aortic valves and walls frequently give rise to murmurs whose presence and significance have not been sufficiently emphasized. Intraventricular murmurs, non-rheumatic in origin, are often present in cardiosclerosis. The cardiosclerotic process prevents coaptation of the mitral cusps and thus produces mitral regurgitation. This murmur is systolic in time and is heard best over the lower precordium; it is usually less loud than the rheumatic mitral regurgitant murmur and is transmitted over a smaller area. Thicken-

ing and dilatation of the aortic walls, common in aortitis, give rise to a loud rough systolic murmur over the right base. As distinguished from aortic stenosis the murmur is less rough and vibrant, and is infrequently propagated along the carotids. If aortic dilatation is marked, the systolic may be followed by a short diastolic murmur, due to regurgitation in the enlarged aortic cavity or to valvular insufficiency.

In some individuals with advanced sclerotic change in the aortic and mitral valves there may be heard a loud systolic murmur over the aortic and mitral areas. Its significance and characteristics with case illustrations are described in Cardio-vascular Clinics.

**Right-sided Murmurs from Auricular Enlargement.**—This murmur has received only occasional mention in the literature. In a few cases of decompensated rheumatic mitral disease which I observed there was a loud rough systolic murmur over the third and fourth right interspaces; in addition a diastolic murmur over the same area was sometimes heard. In all the cases with loud right-sided murmurs which I fluoroscoped, there was enlargement of the right auricular curve, and no enlargement of the aorta. A few illustrative cases are herewith epitomized.

Case I.—Male, age 17, with a definite rheumatic history, had upon hospital admission, the typical physical signs of a double mitral lesion. There was also a visible pulsatile area to the right of the sternum the size of a pigeon's egg, over which was heard a loud rough systolic murmur; a rough palpable thrill was also felt. The patient died after one week. At necropsy, a few pericardial adhesions were found between the cardiac apex and diaphragm. There was 20 cc. of clear fluid in the pericardial sac, the heart weighed 520 gms. The right auricle was hugely dilated (Fig. 268). With the heart *in situ* the right border was 8 c.m. from the midsternal line, the left border, 10 c.m. from the same line. The greatest thickness of the right ventricle measured 1.2 c.m., the left, 1.8 c. m. The right auricle was irregularly circular in outline and was tremendously distended, well shown by distension of the veins of the auricle, a few of which were the size of ordinary lead pencils. The wall of the right auricle was twice as thick as the normal. The right auricular appendix was also enlarged and distended. The tricuspid orifice readily admitted three fingers. There was slight thickening of the tricuspid valve. There was a typical button hole stenosis of the mitral valve.

Case II.—Male, age 38, had chorea as a child and heart trouble for years. Despite this, he was always athletic and active until two years prior to my first examination. He then had his first attack of dyspnœa. A year and one half later he suffered from a pulmonary infarct, hemoptysis and long continued fever. Since then he has been constantly decompensated: The chief symptoms were cough and dyspnœa. Examination: The systolic blood pressure is 85, the diastolic 79. There is marked venous pulsation in the neck. The heart is tremendously enlarged. There is a palpable systolic thrill and a heaving impulse over the lower half of the left chest. Over the right base

there is a somewhat rough systolic murmur transmitted to the aorta in the jugulum. At the apex, a double mitral murmur is heard. The presystolic is heard only at the apex while the systolic is transmitted to the axilla, and to the left sternal border. To the right of the sternum, in the third and fourth interspaces, a loud systolic and a loud diastolic murmur are heard; both are propagated toward the right axilla and the epigastrium. The fluoroscope showed an enlarged right auricle, a normal sized aorta and left ventricular hypertrophy.

Case III.—Female, married, aged 26, had had several attacks of hemoptyses for three years. There has been dyspnœa and edema of the legs for over one year. On examination, the patient is quite cyanotic. The typical signs of a double mitral lesion are heard at the apex. To the right of the lower half of the sternum, there is a rough systolic murmur which is transmitted somewhat to the left.

It is apparent that the auscultatory signs of assumed right auricular enlargement are not always alike. Whatever its time relationship, however, the murmur is always rough and loud. It may be systolic alone or systolic and diastolic. Sometimes a systolic palpable thrill is also present. The systolic murmur is heard best in the third and fourth interspaces, immediately to the right of the sternum and is transmitted to the mid-sternum and to the right nipple region. I conceive the systolic murmur as due to tricuspid regurgitation through an extremely dilated ring into an enlarged auricular chamber, hence the transference of the tricuspid murmur from its usual site. It is significant that liver pulsations were absent in my cases, perhaps because the regurgitation spent itself in an upward (auricular) direction rather than toward the liver. The mechanism of the right sided diastolic murmur which was occasionally heard in these individuals seemed similar to that of aortic aneurism, that is, the dilated auricle acted like a dilated aorta, and after ventricular systole ceased, the regurgitated blood in the auricle passed back through the tricuspid valve during diastole. It may be necessary to differentiate the physical signs of right auricular enlargement from those of aortic aneurism, especially when a double murmur is present. This is readily done by the fluoroscope, for in right auricular enlargement, aortic pulsation is practically normal. Besides, the usual physical signs are present at the right base (first and second right interspaces) in cases of aortic aneurism.

**Intracardiac Murmurs of Non-organic Origin.**—Murmurs arising in the heart and not due to organic disease have received many names and have been ascribed to various causes. They have been termed accidental murmurs, hemic murmurs, adventitious sounds and functional murmurs. They have been ascribed to anemia, to relative valvular insufficiency (incompetency) allowing leakage and regurgitation of blood; and to return of the blood in the large venous trunks. It is apparent that much confusion exists regarding both the terminology and the etiology of these intracardiac murmurs. In the description to be given, I shall apply the term "functional murmur" to those

adventitious sounds in which no organic cardiac lesion is assumed to be present.

Functional cardiac murmurs are very common. The fact that they can occur without cardiac disease and without producing symptoms, requires emphasis. They are often merely accidental discoveries in the routine physical examination. The presence of these murmurs in healthy people as well as in such conditions as fever and anemia, suggests a varying etiology. The usual chief characteristics of non-organic murmurs are their softness and limitation to small areas. There are numerous exceptions, however. The murmur may be rather loud, somewhat rough, and when found in the lower precordium, may be slightly transmitted above or to the left. These exceptions are especially frequent in children and young adults. The most common site of the functional murmur is at the apex. It is systolic in time and accompanies the first sound, usually as a soft whiff. In addition to their presence in normal individuals, such functional murmurs are found in patients with rapid heart action, in those with flabby general musculature, and in those with anemia. In anemic individuals, the changed character of the blood is usually assumed as the etiological factor. This hypothesis, however, is not based upon any experimental data. In those with flabby musculature, the cause seems to be dilatation of the musculature supporting the mitral ring, with consequent leakage and regurgitation; this is commonly termed relative insufficiency or incompetency of the mitral valves. Perhaps many of the murmurs in anemia (so-called hemic murmurs) heard in this location have the same etiology, for grave anemia conduces to lack of proper muscular tone. In tachycardia, improper and faulty closure of the mitral cusps, alone or in addition to relative mitral insufficiency, may be the cause of this functional murmur at the apex.

A source of diagnostic confusion sometimes exists in differentiating the non-organic intracardiac murmur heard over the apex from the organic murmur of mitral insufficiency. The distinction between typical non-organic and typical mitral organic regurgitant murmurs is not difficult; the latter are rougher, louder and are heard and transmitted over larger areas than the former. The second pulmonic sound is apt to be accentuated as compared with the second aortic. When the characteristics of the two overlap, as, for example, in rheumatic fever in which the fever itself or the endocarditis may cause the murmur, the distinction between the organic and functional becomes exceedingly difficult. In doubtful cases with fever, examination must be made after the fever has run its course in order to arrive at definite conclusions. In anemia, the association with other "hemic" murmurs, especially systolic murmurs at the right base, aids in the differentiation. The correlation of other data—lack of symptoms of heart disease, general type of muscular development, occupation (sedentary or otherwise), the type of heart as seen fluoroscopically—all these are elements which require consideration in the differential diagnosis between functional and organic murmurs. Non-organic murmurs

are commonly found in patients of sedentary habits, with flabby musculature and ptosed abdominal organs. The distinction in the middle-aged and elderly between a mitral inorganic (functional) murmur and that found associated with myocarditis and cardio-sclerosis is of extreme importance. The differentiation must be based upon the usual characteristics of the non-organic murmur. If this be soft and blowing, and be not transmitted, one may infer that the mitral ring is dilated and that the murmur is of functional origin. The diagnosis of myocarditis must then be predicated upon the usual evidence of myocardial insufficiency, and upon the presence of an intra-ventricular murmur (q. v.) characteristic of cardiosclerosis (Chapter XVII).

Aberrant fibers coursing through the ventricle, formerly termed aberrant tendons, can presumably give rise to loud systolic murmurs over the precordium. There seems to be no method of distinguishing these from other non-organic or organic murmurs. How often such murmurs occur is not known; they would seem to be extremely rare however. The tricuspid murmur of relative insufficiency may occur with decompensation from any cause that produces venous congestion—emphysema, myocarditis, endocarditis, etc. Indeed the murmur may itself be regarded as an evidence of an inefficient circulation, since it is due to interference with venous return flow in the pulmonary circuit to the right heart. This tricuspid murmur is of varying intensity, is heard over the lower sternum and is usually transmitted to the right; an enlarged pulsating liver is occasionally present. Sometimes the liver pulsates in the absence of a tricuspid murmur. Since this could scarcely occur without regurgitation, it is clear that the latter may be present without producing a murmur. The frequency of relative tricuspid insufficiency without valvular disease is added evidence that mitral regurgitation with its murmurs may also be due to relative valvular insufficiency and to abnormal dilatation of the mitral ring.

A non-organic, soft, blowing systolic murmur is frequently heard over the aorta, particularly in anemia. It is also heard occasionally in the overacting hearts of young individuals. It has none of the characteristics of aortic stenosis, however (q. v.). A similar murmur over the aorta is found in decompensated mitral lesions. I have occasionally heard a short diastolic aortic murmur in mitral lesions in the young; it has none of the clinical characteristics of aortic regurgitation. It is usually temporary and disappears within a few days. Perhaps mechanical torsion or tension on the aortic ring with consequent regurgitation is a factor in its production.

**Extracardiac Non-organic Murmurs.**—Pericarditis is not here included. We have just described functional murmurs due to intracardiac disturbances. We shall now discuss non-organic murmurs of extracardiac origin. The most common of these is the so-called cardio-respiratory or cardio-pulmonary murmur. It is usually soft and blowing in character, systolic in time, and best heard over the left base. Occasionally these characteristics change. Thus the murmur may be rough and loud, and be transmitted along the left

sternal border and lower precordium as a somewhat superficial, squeaky sound, resembling a friction sound. The cardio-respiratory murmur is ascribed to compression of lung tissue between the heart and chest wall. During inspiration, the lung covers a large part of the root of the pulmonary artery and the upper precordial surface, facts which can be corroborated by fluoroscopic examination. This also explains why the murmur is usually heard best when the lung becomes inflated at the end of inspiration. However, it is sometimes loudest at the end of expiration, possibly because in some patients the pad of lung between the chest wall and the heart is abnormally large, thus diminishing the transmission of the murmur. The murmur is found frequently in children. It is not uncommon in frail women with thin chest walls. The fact that the intensity of the murmur depends upon respiratory phases and that it is never accompanied by a palpable thrill aids in distinguishing it from other types of organic cardiac murmurs heard in this area.

**Reduplicated Sounds and Reduplicated Apical Impulses.**—These reduplications represent again a distinct type of adventitious sounds. They have received various names such as split sounds, gallop, canter, and triple rhythm, and "bruit de rappel." The term "reduplication" seems preferable because though many of its etiological factors are still obscure and problematical, it conveys some concrete idea of the underlying phenomenom, namely a doubling of a sound.

Reduplicated second sounds are of valvular origin and can usually be clearly defined by ausculation. In the order of their frequency, reduplicated second sounds are heard at the apex alone; both at the apex and right base, and lastly over the pulmonary artery alone. The time of their occurrence in the cardiac cycle and the study of electrocardiographic sound records demonstrate that they are due to closure of the semilunar valves. Their presence in various positions and combinations suggest differing etiology. In many instances conclusions regarding the cause can for the present be only tentative. One known cause of reduplicated second sounds however is asynchronous closure of the pulmonic and aortic valves. In this type the elements constituting the reduplication are heard very close to each other. Reduplicated second sounds may also be due to asynchronous closure of the individual leaflets in either artery. I saw an excellent example of this in a case of a bulging aneurism of the arch of the aorta in which one could feel as well as hear the distinct and seperate closure of the semilunar valves. In other types, the sound elements may be separated by a distinct hiatus. In these, the first part is undoubtedly due to closure of the semilunar valves, since it occurs in the cardiac cycle in early diastole soon after the A-V valves open. The second part of the reduplication has been ascribed to a process somewhat similar to the causation of the third heart sound, that is, to quick ventricular filling with resultant vibration of the floating cusps. This explanation, however, seems untenable, for the reduplicating elements seem identical on auscultation and have been so proven by graphic sound records. As a more plausible

explanation, it appears to me that a sharp systolic wave can produce a secondary reflux one, which, impingeing upon the already closed and tense semilunar valves, sets these in vibration and thus causes the second element of the reduplication. This would account for its frequent presence in tachycardia, in mitral stenosis, and over the great vessels at the base of the heart.

Reduplicated *first* sounds are usually classified under "presystolic gallop rhythm." The term—presystolic—is inexact, however, for the first element alone of the reduplication is presystolic. Under reduplicated first sounds—a preferable name—I wish to include only those that are not accompanied by reduplicated apical impulses, from which, as I shall point out, they are etiologically distinct. Thus limited, reduplicated first sounds (*i.e.*, without reduplicated apical impulses) are heard only in the apical region. It can be demonstrated by simultaneous electrocardiographic sound records and electrocardiograms that the first element of this type of reduplicated first apical sound actually lies in presystole, and hence must be due to some presystolic event in the cardiac cycle. Most probably this is auricular contraction. The occurrence of such reduplication may depend upon a hypertrophied or overacting auricle.

**Reduplicated Apical Impulse.**—This type, always accompanied by a reduplicated first sound but of different origin from the above, is found especially in conjunction with hypertrophied left ventricles and in aortic disease. It is occasionally found in the overacting normal heart. Upon auscultation, besides the double first sound, there is a distinct sense of a double shock or impulse, often evident upon palpation alone. Since the reduplicated apical impulse has been found in lesions of the main branch of the auriculo-ventricular bundle, ventricular asynchronism is evidently one of its causes. The clinical diagnosis is here aided by the fact that bundle lesions are usually accompanied by signs of myocarditis and by regular slow pulse rates between 50 and 60 per minute.

In addition to patients with bundle lesions, I have observed identical double apical impulses in those without electrocardiographic or other indication of bundle lesions. In several such instances there was definite fluoroscopic evidence that the double impact was due to a secondary ventricular movement following the primary systole. The exact cause of this double impact I could not definitely determine. In an effort to seek its explanation, the following must be recalled. It is known that the normal first sound is composed of three elements, first: Tension of the auriculo-ventricular valves; secondly, the muscular element, ventricular contraction; and thirdly, ventricular impact against the chest wall. The first element of the reduplicated sound under discussion is probably due to these usual three normal factors just enumerated. Regarding the second part of this reduplication it is difficult to state to what extent and proportion these three factors are concerned. To return to the fluoroscopic examination of patients with the reduplication, one may observe not only the ventricular systole but,

what may be termed "ventricular fling," seemingly comprising different areas. Sometimes this involves the apex alone, at others, the entire left ventricle seems to take part in the second movement. It is impossible to determine how far such ventricular fling resembles normal physiological systolic contraction. It may, for example, represent an attempt to regain ventricular tone, an effort interfered with by ventricular distension from varying amounts of residual blood following systole. In general, it seems in harmony with clinical observations to seek mechanical intraventricular causes for the reduplicated apical impulse. A weakened myocardium may fail to properly empty the overdistended ventricle, a condition favorable to the production of a double impulse and reduplicated first sound. A similar condition can occur in patients with long continued fever, as in typhoid when myocardial weakness may be only temporary and functional. Again, it can occur in those suffering from myocarditis and resultant myocardial insufficiency. This was well illustrated in an instance of alcoholic myocarditis in which at necropsy the ventricles were found hypertrophied and riddled with scar tissue. A reduplicated apical impulse was present during the stage of decompensation alone; the electrocardiogram showed a deep M complex (Chapter IX). When decompensation was temporarily restored, the double apical impulse and M complex disappeared, the ventricular complex assumed a normal outline.

Reduplicated apical impulses are frequently present in aortic regurgitation. In these cases they may be directly due to the blood which regurgitates into the ventricle; this then produces a ventricular shock which becomes audible as well as palpable at the apex. A double apical impulse is also found in left ventricular hypertrophy with hypertension. The violence of ventricular action and the impact of residual blood may here account for the second element of the reduplication.

**Physical Signs of Ventricular Hypertrophy.**—The possibility of correctly diagnosing cardiac hypertrophy by the usual physical signs depends chiefly upon the extent of the hypertrophy and the thickness of the chest wall. For example, the tremendous impact to the thoracic wall in the far advanced and extreme ventricular hypertrophy of typical aortic insufficiency can scarcely be mistaken, for it produces a visible and palpable shock to the entire left half of the chest. It is however the minor degrees of cardiac hypertrophy which require diagnostic consideration. Hypertrophic and enlarged hearts in which myocarditis is an important pathological factor, are often not diagnosed as enlargements because the impaired condition of the circulation is such that the usual vigorous, unhampered action indicative of hypertrophy is absent. Indeed, as has already been pointed out, if the diagnosis can be made by other means (electrocardiograms and the X-ray) a weakened impulse is often a sign of weakened myocardium. The diagnosis of hypertrophy must then depend upon the data already indicated in the description of the physical signs of myocarditis (Chapter XVII).

**Left ventricular hypertrophy,** when typical, produces a booming, loud first sound over the entire cardiac area, and a broad, heaving systolic impact distinctly palpable well outside the normal cardiac limits. It must be remembered, however, that these characteristics may be simulated in thin-chested individuals in whom the heart is well applied to the chest wall, or by hearts in which the systolic action is violent (for example, in exophthalmic goiter). Such instances demonstrate the necessity for reserve in the diagnosis of ventricular hypertrophy, particularly in view of the fact, already pointed out, that it is extremely difficult to delimit by percussion even the approximate size of the cardiac area. In other words, the physical evidence of cardiac enlargement must be definite and unmistakable before its diagnosis is ventured. An accentuated second sound at the apex or at the second right interspace is sometimes accepted as an indication of left ventricular hypertrophy. It is true that these abnormally accentuated sounds are often found in hypertrophy; however, they must be regarded not as evidence of hypertrophy, but rather of hypertension in which left ventricular hypertrophy is often, but not invariably, present. A reduplicated apical impulse (gallop rhythm) is sometimes present in the hypertensive cases, especially when hypertrophy exists.

**Right Ventricular Hypertrophy.**—Its diagnosis is even more difficult and problematical than that of the left. A second pulmonic sound, relatively or actually accentuated when compared with the second aortic, is regarded as evidence of overstrain of the right heart, and hence indicative of probable right sided hypertrophy. However, I have observed moderate accentuation of the pulmonic second sound very frequently in children and adults with normal hearts in whom tachycardia existed. I believe an accentuated second pulmonic sound is of value as indicative of probable impairment of the right ventricle only if the accentuation is pronounced and if tachycardia is absent. Even then the question of hypertrophy must rest upon the length of time during which the accentuation has been observed, upon the type of lesion, and upon other associated data. In marked right ventricular hypertrophy the apex may be pushed to the left so that the anterior surface of the heart consists chiefly or entirely of the right ventricle. Furthermore, the cardiac area of dullness may not be increased to the right because the right ventricle normally lies upon the diaphragm. Marked visible and palpable epigastric pulsation and systolic retraction of the apical impulse are the physical signs upon which most stress is laid in the diagnosis of right ventricular hypertrophy. In a series of cases in which the ventricles were weighed at necropsy, Lewis did not find either visible or palpable epigastric pulsation or thrust in a sufficient percentage of cases to make these signs of diagnostic value. These are examples of the difficulties with which the diagnosis of right ventricular hypertrophy is surrounded.

It is thus evident that, except when massive, the clinical diagnosis of left or right ventricular hypertrophy from physical signs alone is often problem-

atical, and that in many instances its presence can only be correctly inferred in conjuction with the known pathological lesion—valvular, myocardial or nephritic.

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## CHAPTER XIV

## GENERAL SIGNS AND SYMPTOMS OF CARDIAC DECOMPENSATION IN ORGANIC CARDIAC DISEASE—THE NEUROTIC ELEMENT IN ORGANIC CARDIOVASCULAR DISEASE—CARDIAC STRAIN—FUNCTIONAL EFFICIENCY TESTS

Before proceeding to the special symptomatology affecting the cardiac musculature and the various valves (Chapters XV–XVII), it is essential to have a broad aspect of the symptomatology of heart disease in general, but particularly of its most prominent symptom—heart failure. Various other terms are used at times instead of "heart failure" such as decompensation, myocardial insufficiency, incompetency or inefficiency. They actually represent indefinable differences in degree rather than in kind; hence I shall not attempt to distinguish between them but shall use them interchangeably. The terminology that includes the muscle element has this decided advantage that it stresses and emphasizes the prime factor in every case of heart failure, namely—the heart muscle—for it is essentially improper performance of, and interference with the heart muscle function or functions, no matter how brought about, that cause myocardial insufficiency.

When describing physiological considerations (Chapter III), the fact was emphasized that cardiac reserve depends fundamentally upon the ability of the heart muscle to properly contract and expand, that is to say, upon the adequate shortening and lengthening of the cardiac fibers. Like other organs of the body the normal heart has a large margin of safety, a large functional reserve. Depending upon the severity and nature of the cardiac disease, this margin of safety is diminished to a varying degree; indeed, a point may be reached at which the heart carries on the circulation without any reserve in store. This means that cardiac activity even though inadequate and inefficient, proceeds at maximal capacity for that individual. As one corollary, it is important for the observer not to be entirely engrossed in minute physical signs and symptoms, but to study the heart from the broad aspect of function and dysfunction; for it is apparent that a heart crippled in function and carrying on the circulation always with a maximal effort will react differently to effort, to recrudesence of disease and to cardiac "insults" than one which, although diseased, carries on the circulation adequately and with sufficient reserve.

**Symptoms of Cardiac Failure.**—Breathlessness and dyspnœa constitute the first indications of decompensation in the vast majority of cases of cardiac disease. Naturally careful and detailed inquiry must be made

(Chapter XIII) as to the type and degree of exercise that first bring on these symptoms. The usual complaint is that climbing stairs or walking against the wind is most apt to cause breathlessness or dyspnœa. It is difficult to divide the latter into stages corresponding to definite entities in cardiovascular pathology, for dyspnœa may change or vary with such fleeting influences as fever, nervous excitement, acute inflammatory disturbances within the heart, etc. It is important to remember that breathlessness may be the result of other conditions, especially pulmonary and renal disease, severe anemia, recovery from a severe acute illness or operation, and that it is also frequently present in cardiac neurosis (Chapter XVII). Painful extra-cardiac conditions (*e.g.*, pericarditis, intercostal myalgia) may also cause rapid breathing. Hence, alone, dyspnœa is not diagnostic or characteristic of cardiac disease; other corrobative evidence is required before the diagnosis of heart disease can be made.

The fundamental causes of various types of dyspnœa first require discussion. Simple breathlessness is not accompanied by any marked chemical changes in the blood.

Dyspnœa is basically due to improper gas exchange in the tissues. This causes a change in metabolism, an alteration which can sometimes be discovered and measured by chemical examination of the blood. It is chiefly through the latter that our knowledge of dyspnœa has been advanced. Abnormal products, or abnormal amounts of normal products in the blood that cause diminished blood alkalinity (so-called acidosis), have been found. Experiments show that alkalies injected into the blood stream cause apnœa (lessened breathing) and that very small quantities of acids and of acid salts, similarly injected, produce hyperpnœa (accelerated and deepened breathing). Carbon dioxide, the chief product of metabolism, acts chemically in this respect like a mineral or organic acid; it causes an increase in hydrogen ions in the blood; these ions represent the chemical factor which measures "acidosis." The respiratory center is quite sensitive to changes in blood reaction, hence the various types and degrees of abnormal breathing—breathlessness, dyspnœa, etc. The mechanism of regulation of the respiratory center seems somewhat as follows: "Acidosis" (diminished blood alkalinity) stimulates the respiratory center to increased activity. With increased activity, there is increased pulmonary ventilation, and consequent reduction in carbon dioxide. This again tends to keep the hydrogen ion concentration of the blood at a normal level. Thus there is a nice mechanism between blood "acidosis," excitation of the respiratory center and increased breathing, a mechanism which tends to diminish the degree of "acidosis" by ridding the body primarily of its increased carbon dioxide. Lack of oxygen (anoxemia), a factor insufficiently known or studied, may possibly exert an as yet unknown influence in the production of dyspnœa. There are several methods of estimating the carbon dioxide content of the blood. It may be measured directly by examining the blood withdrawn from a vein (Van Slyke method);

or it may be estimated indirectly by various apparatuses for determining the carbon dioxide content and tension of the alveolar air. It will be shown that conclusions drawn from such examinations bear an important relation to cardiac dyspnœa.

Another method of studying dyspnœa is the simpler clinical one of direct observation of the rate and depth of breathing; this naturally gives the measure of pulmonary ventilation. As an instrumental method (a calibrated recording spirometer) Peabody has recorded not only the rate and depth of breathing, but also measured the minute-volume of respiration. Instead of using exercise to produce dyspnœa, he had the patients breathe air containing increasing amounts of carbon dioxide, the chief factor in the production of dyspnœa. It was found that the severely decompensated cardiac cases could stand but little increase in the amount of carbon dioxide as compared with the normal control. The abnormal *rate* of respiration was about the same as the normals, but the *depth* of respiration was only about half the latter. Clinically, this means that the cardiac patients could not ventilate their blood sufficiently to overcome the increasing amounts of carbon dioxide by deeper breathing; hence, their blood remained saturated with carbon dioxide and the respiratory center remained abnormally stimulated. Stated in another fashion, the pulmonary reserve of decompensated cardiac cases is considerably below the normal, a result to be expected, and comparable to their diminished cardiac reserve. Peabody also studied the vital capacity, the maximum amount of expired air following a maximum inspiration of normal individuals and those with cardiac disease. He found that in a general way lessened vital capacity ran parallel with the tendency toward or the actual presence of cardiac failure. Observations of vital capacity may therefore be a ready objective method for noting degrees of heart failure.

It should be remarked that dyspnœa occurring with heart failure may be composed of several associated factors, such as cyanosis, emphysema, pulmonary stasis, congestion and hydrothorax, all perhaps previously due to the heart affection, yet any one of which may in individual instances, overshadow the heart itself as the cause of dyspnœa. Indeed, mechanical blocking of pulmonary ventilation by emphysema etc., may of itself cause lessened vital capacity. As with all instrumental methods, so here it is in addition always necessary to examine the patient carefully in order to gauge results properly.

A factor in dyspnœa perhaps insufficiently recognized is the result of functional disturbance of the respiratory centers. This sometimes produces rapid and shallow breathing—tachypnea, a condition found especially in those with cardiac neurosis (Chapter XVIII).

**Cheyne-Stokes Breathing—Periodic Respiration.**—Periods of gradual increasing dyspnœa followed by periods of apnœa are sometimes found in the late stages of cardiac failure. Its periodicity may perhaps resemble etiologically a peculiar type of breathing (the so-called Biot type) which is occasionally found in patients with advanced arterio-sclerosis and extreme hypertension.

There are sudden attacks of air hunger with deep respiration (hyperpnœa) followed by a phase of absence of breathing (apnœa). Its etiology probably depends upon extreme, sudden differences in the amount of oxygen and carbon dioxide in the blood of the cerebral vessels, combined with localized cerebral endarteritis. The apnœic period would then be due to cerebral anemia and lack of oxygen (anoxemia); the hyperpnœa, to massive accumulation of carbon dioxide in the respiratory center. During apnœa, carbon dioxide saturates the blood. This excites the center to increased activity: Hyperpnœa and increased pulmonary ventilation results, with consequent riddance of the excess carbon dioxide. Again, with a lowered carbon dioxide, overstimulation of the respiratory center ceases and the apnœic period begins.

Attacks of sudden, almost paroxysmal dyspnœa, often nocturnal in character, are not uncommon in heart disease. When unattended by the physical signs of pulmonary stasis or edema, or when there is no evidence of sudden, added cardiac damage, such paroxysms of dyspnœa are difficult to explain. They may perhaps depend upon varying degrees of dilatation in an already crippled heart, thus interfering with proper propulsion of the blood in the systemic and pulmonary circuits. Such assumed attacks of cardiac dilatation, however are not diagnosable by our present methods of examination (Chapter VI). Hence, this assumption can be based only upon negative findings at the time of examination and upon the probability that cardiac dilatation can and does produce dyspnœa.

Orthopnœa may be defined as an extreme continued state of breathlessness in which the individual must constantly sit up and bring into play all the accessory muscles of respiration. When due to the heart alone and not paroxysmal in nature, orthopnœa is a sign of extreme decompensation.

All types of dyspnœa are naturally increased by exercise, for then a greater demand is made upon the circulation than when at rest. That vaso-motor influences can have a perturbing effect upon dyspnœa especially in those with hypertension and cardio-sclerosis is evidenced by the fact that dyspnœa is sometimes considerably increased when patients walk or even drive in the open air, yet these individuals are perhaps fairly comfortable when walking indoors. I believe this is only explicable upon the supposition that the colder open air causes contraction of the skin arterioles with consequent sudden filling of the venous reservoirs and capillaries.

Having described the various fundamental patho-physiological causes of dyspnœa, it should again be emphasized that dyspnœa—abnormal shortness of breath—is the most common clinical and obvious sign of beginning heart failure and that in a general way its degree is roughly parallel to the degree of heart failure.

Cough is another common symptom of cardiac failure, and, like dyspnœa, may be due to various causes, hence it too requires careful observation and study. Cough may be purely reflex in character. This is the true so-called "cardiac cough," and is extremely difficult to differentiate from a purely

neurotic cough. Such differentiation can only be made upon the basis of a careful physical examination not only of the heart but also of the lungs, in order to exclude bronchitis and evidence of visceral congestion (q. v.). "Cardiac cough" is dry and hacking in character and is usually accompanied by slight dyspnœa. Its origin seems due to reflex irritation of the nerve supply of the bronchial tree, the impulse probably passing by way of the ganglia surrounding the root of the aorta and the bifurcation of the trachea (Chapter III).

There are other types of cough in heart failure which are due to actual physical changes in the bronchial tubes, lung parenchyma or pleura, or there may be a combination of several of these pathological conditions. A pulmonary infarct, when typical, gives rise to hemoptysis, fever, and more or less extensive pulmonic consolidation, with its attendant physical signs. Sometimes the site of the infarcted area may not be localizable if a central portion of the lung be involved, if pulmonary congestion instead of consolidation be present, or if there be no accompanying dry pleurisy with its typical rales over the infarcted lung.

When cough is due to bronchial congestion, the physical signs are the usual ones of bronchitis. In addition, rather marked sibilant breathing may be present, thus resembling bronchial asthma. The wheezing inspiration, however, is probably caused by the bronchial congestion and mucus in the bronchial tree and not from any anaphylactic reaction. Another type of spasmodic bronchial cough is that found occasionally in luetic aortitis (Chapter XVI).

"Cardiac asthma" is a term difficult to define, for different writers place it in entirely different etiological categories, such as dyspnœa, pulmonary edema, bronchitis, etc. In its strictest sense it applies to the type of nocturnal paroxysmal dyspnœa already described (q.v.). Hence, the term "cardiac asthma" unless properly characterized and defined is too generic to be of clinical use.

**Sudden pulmonary edema,** always a threatening complication, gives rise to soft, bubbling, mucous rales usually over the entire chest. Extrasystoles or complete irregularity of the pulse (auricular fibrillation) is apt to accompany the attack. Limiting ourselves to cardiac causes and not to such extracardiac factors as toxemia, drugs, poisons, etc., the etiology of acute pulmonary edema is by no means always clear. When due, for example, to a resolving pneumonia following an infarct, the cause is sufficiently manifest. This however does not explain the usual attacks of sudden acute pulmonary edema. Although our knowledge of pulmonary edema is still inadequate, the prime factor seems due to some influence which markedly and suddenly diminishes the ventricular output of one chamber as compared with its fellow. Perhaps cardiac dilatation, especially when unequal in the two chambers, may represent such a condition. As already pointed out, for the present the diagnosis of cardiac dilatation can be based only upon

surmise and questionable clinical criteria, for such dilatations do not give rise to unequivocal physical signs. What seems to me a much more probable cause of acute pulmonary edema is sudden occlusion of the smaller or larger coronary branches (Chapter XXIII) by thrombosis or embolism, the pulmonary edema immediately overshadowing the dyspnœa, precordial pains and usual prodromata of coronary occlusion. Such etiology of pulmonary œdema is further borne out by the clinical fact that it is especially frequent in cardiosclerosis with hypertension, a complex in which coronary disease is very common. Pulmonary edema is rarely the first sign of cardiac failure unless it has followed severe cardiac overstrains or some sudden neurogenic "insult" such as fright or shock.

**Cyanosis.**—It is a common clinical observation that cyanosis, often a prominent and obvious sign of advanced or long standing valvular disease, is not necessarily accompanied by marked dyspnœa. On the other hand, patients of the cardio-sclerotic type (Chapter XVII), especially those with renal complications, are very apt to be dyspnœic but not cyanotic; indeed, they may be decidedly pale. Nephritic dyspnœa is often nocturnal; it may then appear suddenly or in paroxysms. It is thus evident that dyspnœa is present in two different groups of cardiac patients: In those who are pale and those with cyanosis. In the cyanotic group, chemical examination of the blood shows no abnormal increase of non-volatile acids; the carbon dioxide tension of the alveolar air is usually normal; there is no or but slight decrease of blood alkalinity (that is, there is no acidosis). In the anemic group, renal complications are frequent, the blood pressure often high, and venous congestion is, as a rule, absent; the carbon dioxide tension of the blood and of the alveolar air is decreased; the acid products, increased (acidosis); both changes may be extremely marked. Such patients often have uremic manifestations. At present, the belief is that these abnormal products in the blood are due, not to overproduction, but to retention from renal insufficiency.

According to recent studies, the cause of cyanosis seems to be deficient oxygen saturation of the blood (anoxemia). In heart disease several factors may play a role in causing the anoxemia—disturbance of the arterial circulation; venous insaturation from congestion; or disturbance of the capillary circulation.

With marked or only slight dyspnœa, cyanosis is most strikingly seen in chronic uncomplicated valvular disease. Unless renal complications are present, the tension of the alveolar air and the blood alkalinity are within normal limits. The dyspnœa, when present, is due chiefly to venous engorgment, the result of myocardial insufficiency. This venous stasis affecting the pulmonary circulation produces deficient oxygenation, which finally results in cyanosis. Cyanosis without dyspnœa is most frequent in chronic decompensated mitral lesions. When the patients are resting quietly, dyspnœa is scarcely evident. It should be pointed out that cyanosis shows

itself at first in turgescence of the mucous membranes of the lips, cheeks, and conjunctivæ; later of the extremities.

Visceral congestion varies considerably in the different valvular lesions. Congestion of the bronchial tree is shown at first by symptoms and physical signs of bronchitis; in the more advanced cases, by pulmonary congestion and hypostatic pneumonia. Mitral stenosis is especially apt to be accompanied by hemoptysis, usually small in amount. Slight febrile changes may accompany the bleeding. Pleural transudates, especially right sided hydrothorax, may be present; this preference for the right side has as yet not been satisfactorily explained. The presumption is that it is caused by the pressure of the right heart on the azygos veins. The liver may be slightly or tremendously enlarged and may even reach the pelvic brim. The enlargement may be permanent or its size may return to the normal when compensation is restored. The shape of the engorged liver is globular; its surface, smooth. If hepatic congestion is a gradual process, there is little or no tenderness on pressure; with sudden decompensation and consequent rapid engorgement of the organ, liver tenderness is marked. The usual sites of tenderness are the epigastrium and the gall-bladder region. Hepatic congestion may be an isolated phenomenon, other organs being spared. It would seem that in such cases the liver tissue acts almost like a sponge, swelling with the venous engorgement. The clinical syndromes in which this condition is apt to occur are mitral disease with the sudden onset of auricular fibrillation, and occlusion of one main coronary, usually the right. Jaundice sometimes accompanies marked hepatic enlargement and is occasionally extreme. In one instance which came to my knowledge, marked destruction of the liver parenchyma was found at autopsy.

The spleen is often found enlarged and congested at autopsy in chronic valvular disease. This enlargement often escapes diagnosis during life, for the edge of the organ is rarely palpable, and evidence derived from percussion alone is untrustworthy. Splenic infarcts may be found in those suffering from bacterial endocarditis (Chapter XV): During life the infarct may be diagnosed by tenderness and pain over the spleen and by a palpable enlargement.

**Edema.**—It is pertinent to inquire into some of the etiological factors concerned in the edema of heart failure because of the complex and intricate nature which modern conception assumes in its causation. In the discussion of dyspnœa it has already been shown how heart failure causes "acidosis." Cardiac failure also causes disturbance of venous and capillary pressure; as a corollary it also disturbs pressure relations in the lymphatics.

Chemically, the edematous fluid resembles a transudate. It is clear, watery, of low specific gravity (around 1005) and contains organic and inorganic salts. It is the abnormal accumulation of this fluid in the intercellular spaces (or serous cavities) which constitutes edema. The formation of edema is closely bound up with the flow of lymph. Normally, there is a

nice balance maintained between arterial, venous, capillary and lymph flow so that there is never any leakage or seepage of lymph during health. With the occurrence of heart failure, this balance is disturbed by certain hydrostatic and physico-chemical factors. Briefly, these physico-chemical factors are diffusion, a movement of molecules from areas of greater to those of less concentration; transpiration, a movement of molecules through membranes, (here applied to the cellular walls of lymphatics and capillaries); osmosis, a movement of molecules through membranes permeable to them but not to the dissolved substances. Colloidal imbibition of water (probably a minor factor), and the influence of chlorides and of the amount of fluid are other factors. In order that any or all of these physico-chemical factors be brought into play, greater hydrostatic forces must be considered, just as in the flow of water. These are first, the "pump" (the heart), the source of energy which determines the rate and volume of blood (and lymph) flow; and second, the "head" of the flow, that is, the difference in potential between the various areas involved. Thus it may be seen how, in heart failure, all these factors may cause complicated interaction; that individual factors may assume varying proportions in different types of heart failure; for example, where kidney congestion plays a role, chemical factors may predominate because the renal filtering function is interfered with; and finally, since we possess only incomplete knowledge, and the actual changes are hidden from view, often broad clinical knowledge and correlation alone can help in the solution of the etiology of the edema.

Edema itself is easily recognized by the well known pitting on pressure: The depressed skin can be readily seen and felt. In pure cardiac failure, the feet and ankles first become edematous; in more advanced cases, there is also edema of the legs, thighs, scrotum and of the abdominal walls; finally, all the subcutaneous tissues and serous cavities become edematous (general anasarca). In the edema of pure nephritis, the eyelids and face first show the edema, later the ankles; in advanced parenchymatous nephritis, general anasarca may also result.

Renal involvement in valvular heart failure ranges from moderate passive congestion to sclerotic changes in the glomeruli. The former is common; the latter, rare. Embolic renal infarcts are rare in chronic rheumatic endocarditis; fairly common in streptococcus viridans infections. Renal congestion is shown by decrease in the amount of urine, by the presence of albumin and casts, and, in severe cases, by lessened excretion of phenolsulphonephthalein (Chapter XXI).

Cardiac pain, or as I prefer to call it, precordial pain is discussed separately in subsequent chapters (Chapters XVIII, XXIV). I shall here limit myself to the precordial pains in chronic valvular disease. The severity of the pain apparently depends chiefly upon the degree and suddenness of decompensation. Its distribution and character vary from a slight sensation of precordial heaviness to agonizing attacks radiating from the chest to the left shoulder,

left scapula, neck, abdomen, and rarely, to the legs. Sudden dyspnœa and pain may also occur as the result of a profound change in intracardiac circulation from embolus or infarct of the coronary or its branches.

**Cerebral Manifestations.**—Excluding cerebral thromboses and hemorrhage (apoplexy), the majority of the serious cerebral manifestations of heart failure are the result of disturbed cerebral circulation. Where there are such complicating factors as associated kidney lesions with "acidosis," or pulmonary stasis with or without hydrothorax, the tendency toward insufficient cerebral blood supply is present; hence, comparatively slight increase in cardiac decompensation will precipitate cerebral manifestations. Drowsiness, sleepiness, mental languor and torpidity are the beginning stages of the latter. Stupor represents a later stage. In aortic lesions, especially that associated with arteriosclerosis and cardio-sclerosis, there are at times periods sometimes lasting several days in which the patient is in semi-stupor or even coma; the condition seems due to actual slowing of the entire cerebral circulation, partly from weakened cardiac power, partly from narrowed and diseased cerebral vessels. In cardiac failure associated with parenchymatous changes in the kidneys and general edema, various stages of cerebral depression ranging from mild stupor to deep coma are found. Such manifestations seem primarily due to cerebral edema. In some types of cerebral endarteritis with accompaning heart failure, epileptic seizures are the rule. Dizziness and syncope are common cerebral manifestations due to transient cerebral anemia.

**The Neurotic Element in Organic Cardio-vascular Diseases.**—Thus far we have been dealing with, and confining our attention to objective signs of cardiac disease. There is a marked modern tendency to depend too much if not entirely upon such objective phenomena. This tendency has been considerably enhanced by the frequent use of instrumental and laboratory aids, such as the electrocardiograph, polygraph, X-ray, blood pressure instruments, blood chemistry, etc. Practitioners as well as students are only too apt to rely implicitly upon data thus furnished, and to pay insufficient, or perhaps no attention at all to the mental and psychical make-up of the individual, with his reactions and social relationships. The author has frequently stressed the outstanding importance of learning how to apply in the sick room the lessons learned from instruments; in other words, their clinical and practical application. But in addition it is important to understand the patient as an individual. It is just in this phase of clinical medicine that the old master clinicians stood out preeminently. Their vast bedside experience taught them almost intuitively to pick out the neurotic, the hyperexcitable, the phlegmatic; and with a kind word here, a humane expression there, an interest in the commoner complaints, they gained not only a tremendous insight into the mental fabric of their patients but with it a control over their psychic reactions.

Our intensive rush of work and our intensive resort to instrumental and other objective aids, has doubtless hastened, if indeed it has not caused the

popular interest in the various medical cults and fads of our time. We do not sufficiently appreciate the mental anguish, the suffering of the patient, whether he has organic disease or not. The patient feels himself a "sufferer;" this is the phase with which his mind is almost exclusively occupied; while we are wrapped up in diagnosis and think little of therapy, especially in chronic or functional conditions; offer little or no psychological encouragement, and forget that a patient even with a functional complaint actually "suffers." We are too apt to dismiss a functional case as hysterical or neurotic because we discover no objective findings. It is perhaps our own disappointment and our own inability to decipher and translate into terms of actual pathology apparently unfounded complaints that produce in us a certain amount of impatience. This impatience is often reflected in a curt demeanour toward the patient. Indeed the patient's point of view is rarely considered; he is simply regarded as an impersonal "case" of functional disturbance or as being "nervous." The patient, on the other hand, is seldom interested in the diagnosis as such. He is chiefly interested in the length of time he is going to be ill, and in the therapy. Is he very sick? Will he get well? How long will it take him to get well? Will he be able to continue his work? Will he have to stop work entirely? Will he have to go away, and if so, for how long? Such questions are even more important to wage earners who must return to their work at the earliest possible moment. In chronic or incurable cases particularly, the physician sees just the hopeless aspect, forgetting the importance of buoying up the patients morale, so to speak, by either minimizing the incurability of the condition, or by instilling hope that the illness will improve in the course of time. There are after all, but few of us who are brave enough to be told frankly that our case is hopeless or incurable, or that the disease will soon kill us. Thus, the physicians' own neglect has nurtured the hope and faith in cults and fads, for they at least give exalted hope and promise relief from the patient's "suffering." In all this, there is of course this definite warning. Not that instrumental and other methods of diagnosis are to be discarded; on the contrary, they are needed all the more in order that we be as sure as possible in the present state of our knowledge that no actual organic lesion escapes diagnosis. But when a final diagnosis of a functional disorder is made, our task is not ended. We must search for social factors—an unhappy married life, fear, fright, business reverses, etc.—as possible etiological factors for a disturbed psyche, and with it, a cause for the various functional disorders. Such factors if found naturally require their proper social adjustments. We should also instill and fortify these patients with a hope of cure. Drugs, even of questionable value; therapy, such as violet ray to the chest for pain—are not only admissible but are of unquestioned value when, in addition, the patient is mentally stimulated by hope, and can be assured there is no serious pathological background for his complaints. To fortify themselves, it may perhaps be a wise thing for physicians to study, not the cults themselves, but the psychic essentials in them which undoubtedly often lead

to cures or at least relief, especially when "legitimate" scientific medicine fails. Faith, belief, hope, humaneness and sympathy, are the keystones of medical cults and fads. These can undoubtedly be used to our own advantage and to that of our patients, if we have sufficient regard for their importance; for important they are, both in organic and functional disease of the human economy The entire subject merits this special mention because it is an almost neglected field of practical, clinical medicine.

Chapter XVIII discusses the pure neuroses, many of which are illustrated in the Cardio-vascular Clinics. Here I shall confine myself to the Neurotic Element in Organic Cardio-vascular disease.

I do not believe that the role of neurotic and psychic disturbances as initiators of symptoms in those with organic cardio-vascular disease has been as sufficiently emphasized as in those with purely functional neuroses. The soldier's heart, hyperthyroidism are examples of the latter (Chapter XVIII). It not infrequently happens that individuals with heart disease get along fairly comfortably until some untoward psychical disturbance such as fright, initiates symptoms from which it may take patients months to recover, indeed they may never regain their old cardiac stability. We are so apt to be engrossed with physical signs during an examination that we may not lend sufficient attention and importance to minutæ in the history, apparently minor factors which often have an important etiological bearing. I have found that especially in mitral stenotic lesions, mental disturbances can have a markedly deleterious influence. It can not only bring on tachycardia but overforceful cardiac action as well; this indeed can occur with lesions which from the inflammatory and clinical standpoints had long been quiescent and stable. When studying such hearts prior to the period of abnormal excitation, one observes no differences either in their fluoroscopic size, the presystolic murmur, or other physical or clinical characteristics which would single them out as being especially susceptible to nervous influences. It is somewhat surprising to note that even from the psycho-neurotic standpoint these individuals present nothing abnormal or characteristic. They have no psychopathic stigmata, they impress one as having had stable nervous systems prior to the onset of their neurotic cardiac symptoms, and they do not show any characteristic in general physique which would align them with so-called psycho-neurotic types. It is important to observe that in those with mitral stenosis and auricular fibrillation, the effect of a neurotic "insult" is much more marked than in those with rhythmic pulses: Cardiac activity becomes more violent, rapid and irregular, and there are more abortive beats. Indeed, I believe that such individuals may die suddenly as the result of a nerve shock.

I shall give a few illustrative cases showing the effect of the psychic element in mitral stenosis.

R. A. E., female, aged 22, of slight physique, has had "heart trouble" since childhood. She had however, always been able to play normally with

other children, with no limitation of her activities. One year ago her mother died unexpectedly. That same day she had "palpitation" and irregular heart action which has since continued. Physical examination shows a typical mitral stenotic lesion with auricular fibrillation. The cardiac rate varied from 100 to 130 per minute. During her stay of several weeks in the hospital neither rest in bed, digitalis nor bromides had any appreciable influence upon the irregularity. She was not dyspnœic; slow walking did not increase the heart rate nor the subjective feeling of palpitation. The main complaints were palpitation and some limitation of physical activity.

L. K. female, aged 19, had a mild attack of grippe trachitis during which she had a "choking spell;" this frightened her considerably. From the patient's description the spell was apparently due to spasmodic pharyngeal cough. The spell was immediately followed by rapid heart action, later by dyspnœa. Upon hospital admission a typical mitral stenotic lesion was found, there were no signs of decompensation, the heart action was regular, the rate, 90 per minute. There was slight tenderness upon pressure over the precordium. Although the patient complained of dyspnœa, she could breathe comfortably when lying flat. She was given a few doses of bromides and was assured that her heart was not damaged. After two days the patient felt relieved and was allowed out of bed.

This was an instance in which the tachycardia was temporary but the subjective dyspnœa and palpitation continued. Reassurance doubtless played an important part in the "cure."

Mrs. C. W., aged 35, had rheumatism 5 years previously. She had been curetted for an abortion several weeks prior to consulting me. The operation was followed by fever, and articular pains and swellings. When brought to my office she presented all the classical symptoms of severe decompensation; anasarca, dyspnœa, hydrothorax. Physical examination showed a typical mitral stenotic lesion, auricular fibrillation and an enlarged heart. The patient was apparently much pertrubed and frightened during the preliminary examination, in spite of the frequent assurance that there was no cause for worry and that no part of the examination was painful. The next step was fluoroscopy. She was slowly assisted to the apparatus by the nurse and myself, apparently reassured and calm. No sooner did she sit down in the apparatus than she gave one cry, and died within two minutes. Most probably fright was the underlying cause of the sudden death: One can only theorize as to how this factor operated, possibly by the induction of ventricular fibrillation and death.

I shall now cite two other instances, one of which also ended fatally, in order to demonstrate definite correlation between psychic "insults" and grave cardiac symptoms.

I. S., dentist, aged 32, married, had scarlet fever complicated by an attack of articular rheumatism lasting one week, eighteen years ago. The only other infections were mild attacks of influenza, one and two years ago,

respectively. Six years ago he was told by a physician that he had "valvular heart disease." Despite this knowledge he was always physically active and athletic and never had any distinct cardiac symptoms until the onset of his present complaint several months ago. He then had a good deal of family aggravation and disputes which worried him considerably and which resulted in attacks of tachycardia and dyspnœa. He finally developed hemoptyses, usually accompanied by severe left sided precordial pains. Fever was never present. Tubercle bacilli were never found. On examination the systolic blood pressure was 150, the diastole, 50. The mouth, thyroid, and throat were normal. There was slight throbbing of the carotids. There was a well defined diastolic murmur heard best over the left base. There was overaction of the cardiac apex, accompanied by a palpable thrill. A soft systolic murmur was also heard at the apex. The fluoroscopic examination revealed definite overaction of the aorta and moderate enlargement of the heart in a downward direction. The knee reflexes were much exaggerated. The urine was normal. There was nothing of moment in the electrocardiogram.

The case was one of rheumatic aortic regurgitation which had long since reached a quiescent stage. Family nagging however had directly caused tachycardia, as the result of which attacks of pulmonary edema and hemoptysis had occurred, or possibly emboli were thrown into the circulation by the violent cardiac action. At any rate, there was no evidence of any rheumatic endocarditic recurrence.

The further course of the case was interesting. The patient was placed on luminal tablets in doses of 1½ grains. He was sent to the country for the summer and was told to do very little walking and to rest out of doors a good part of the day. At the end of several weeks the slight tachycardia and continued consciousness of his heart action present at the first examination disappeared, he was able to walk at a natural pace without any cardiac symptoms. Recently he has again taken up his dental work and feels as comfortable as he ever did. He is gradually taking up his old routine of life.

A. S., male, aged 68, gives a history of an old gonorrhea, otherwise he has had no other infections. He has had sugar in his urine as high as 1½ per cent. for many years: There was no albumin. There has been increasing dyspnœa for three years. There were no precordial pains. Examination: The systolic blood pressure is 125, the diastole 10. The carotids throb. There is a broad heaving apical impulse. There is no pain on pressure over the heart. The heart is considerably enlarged. On auscultation at the base, there is a slightly accentuated second sound; at the apex the first sound is reduplicated and is accompanied by a systolic murmur. The margin of the liver is at the umbilicus. There is slight edema of the legs. The urine and reflexes are normal. Orthodiascopic examination shows left ventricular hypertrophy and aneurismal enlargement of the first portion of the aorta. The electrocardiogram shows left ventricular preponderance. The Wasserman blood

examination is negative. The diagnosis was cardiosclerosis and left ventricular hypertrophy with moderate decompensation. The patient improved after having been digitalized.

The patient had a very small rectal fissure which caused much pain and which was sensitive even to the slightest touch. He was finally sent to another physician for further rectal examination with the distinct admonition to the physician that undue pain be carefully avoided and that nothing be done or said that would upset, pain or frighten the patient. This warning was heeded, yet the patient was so frightened that he nearly fainted on the examining table. Thereafter, there was immediate aggravation of the cardiac symptoms: Dyspnœa became at first distressing, later constant. The patient died after a few weeks.

The patient represented a case of severe cardiac disease in whom the fatal termination was undoubtedly hastened by the patient's fright at the very trivial and carefully executed rectal examination.

I could multiply instances in which the psychic factor initiated symptoms in otherwise stabilized cardio-vascular disease, but I believe these few cases are sufficiently illustrative. They likewise illustrate why such symptoms as tachycardia and auricular fibrillation are not controlled by our usual remedies in these individuals, and why our prognostic viewpoint regarding them cannot conform to the usual type in which such neurotic factor is absent. In addition to the customary drug remedies, reassurance and other psycho-therapeutic measures play an important part in the treatment.

Nervous upsets in otherwise stabilized cardio-vascular disease seem to act deleteriously in two general directions: First, by way of the vasomotor system; and secondly, by way of the accelerator arc of the sympathetic nervous system. The first effect may, for example, upset the balance in the venous reservoirs of the splanchnic system or of the systemic venous capillaries, thus storing up blood in unusual quantities somewhat similar to what is found in some types of shock (Chapter XXVI). The second effect, that on the accelerators—races the heart unnaturally, and in an already diseased organ, intensifies a tendency to disturbed circulation. Often these two effects are undoubtedly combined. One cannot of course study the effects of deleterious nerve influences objectively or quantitatively, but can only draw inferences from physiology and physiological experiments. The group of clinical cases above described seems best explicable upon such bases.

**Cardiac Overstrain.**—This subject is taken up here in connection with cardiac failure, not because cardiac overstrain is especially frequent in those with organic heart disease—indeed it is infrequent in them—but because the symptoms at least superficially resemble those of heart failure and are often mistaken for the latter. If through undue physical exertion the heart of the patient who already has slight or beginning heart failure be overtaxed, the result is increase and persistence of such symptoms as cough, dyspnœa and tachycardia. In those with quiescent endocardial lesions, some of

whom perhaps have no knowledge of their heart disease, the result of cardiac overstrain is comparable to that found in normal hearts (see below), but the symptoms are longer continued and they react less readily to therapeutic procedures.

It is however in those with normal hearts that the question of cardiac overstrain is of most importance, for in them it occurs most frequently. It is often assumed that cardiac dilatation (Chapter VI) is the basic underlying factor produced by the strain. Unless one wishes to assume that the type of cardiac dilatation accompanying cardiac overstrain in healthy hearts is different, symptomatically and otherwise, from that ordinarily found in cardiac disease, one does not observe in healthy overstrained hearts, such alarming symptoms as pulmonary edema and heart failure. The symptoms and signs most often found are tachycardia, precordial pains, extrasystoles and dyspnœa (q. v. Cardio-vascular Clinics). They are also present in heart failure, hence the frequent confusion with the latter. The importance of the proper diagnosis of cardiac overstrain lies not only in the immediate discomforting symptoms but also in the fact that the patient often suffers for months and even longer with the symptoms common to irritable hearts and cardiac neurosis (Chapter XVIII). Based partly on the future progress of these individuals, and partly on the absence of evidence of actual decompensation, it appears most probable to me that the primary injury in cardiac overstrain in the healthy heart is a neurogenic one, possibly in the form of exhaustion of the normal tone of the controlling nerves of the heart.

**Functional Efficiency Tests.**—These are described in this connection because they have a certain bearing upon the cardiac reserve, and therefore upon heart failure. For years various tests have been in vogue in order to graphically or mathematically estimate the cardiac reserve power, the muscular efficiency, the work force of the heart. The methods employed have been of the most diverse kinds; a few will be briefly mentioned. A fluoroscopic examination is one means which has been employed (Moritz and Dietlen). If orthodiascopic observation reveals increase in the size of the cardiac outline after exercise (*i.e.*, if the heart dilates instead of contracting), it is regarded as a sign of an insufficient heart: If contraction follows exercise, the heart is regarded as efficient. These conclusions have been disputed by others. Differences in systolic blood pressure following exercise, and differences in pulse pressure in the standing and sitting positions, are also employed as guides of cardiac efficiency. An attempt has likewise been made to estimate the muscular efficiency and cardiac load by a standard quotient obtained by dividing the pulse pressure by the systolic pressure. Another formula (Stone), consists in estimating the heart load as the resultant of the division of the pulse pressure by the diastolic;this should normally approximate 50 per cent. Still another test for functional efficiency, a modification of the older Graupner method, consists in the observation of the time required for the blood pressure to return to its level after exercise (Barr-

inger). It has been found that, in normal individuals, the systolic blood pressure is raised very soon after a moderate amount of work; with more severe exercise, the maximum systolic blood pressure is not reached until one or more minutes after exercise has ceased. This is the so-called "delayed rise." This observation has been recently applied in the study of the functional capacity of diseased hearts; instead of an ergometer, the foot pounds of exercise or work are computed by the use of dumbbells and bars of various weights; these are lowered and raised a certain number of times to known heights. A criticism of this procedure (Rapport) shows that if blood pressure readings be taken *immediately* after exercise, there is at first a preliminary fall of pressure. Since however such a preliminary fall is almost constant, it may not invalidate clinical conclusions if observations be made according to the Barringer method. Another more applicable method is to have the patient climb a certain number of steps and then observe what effect the procedure has had upon the blood pressure (Wilson). The blood-pressure methods of studying functional capacity seem especially applicable to those with cardiac neurosis in whom breathlessness, dyspnœa and tachynpnœa are prominent symptoms, yet whose functional cardiac capacity may be found normal according to these blood-pressure tests. Thus, one may perhaps be assured of the muscular efficiency of such a heart, despite somewhat alarming symptoms.

It has already been shown that cardiac dyspnœa depends upon the vital capacity of the lung and upon the "pulmonary reserve." These factors may perhaps be readily employed as indicators of cardiac efficiency. In those with healthy or compensated hearts, the pulmonary reserve is ample; in those with decompensation from various causes, it is considerably decreased. The instrument employed is a specially calibrated spirometer with a recording kymograph attachment. The patient breathes through a rubber mouth-piece connected with the spirometer. It seems possible that minute-volume estimations may give quick and reliable information of a tendency toward dyspnœa and thus the instrument may be used as a clinical indicator of the functional capacity of the heart.

Among other functional tests are the following. The **Katzenstein test:** Here, the heart work is increased by compressing the femoral arteries from two to five minutes. The normal reaction found in healthy and compensated hearts consists in a rise of the brachial blood pressure, of from 5 to 15 m.m., the pulse rate falls. In cardiac failure, the blood pressure falls and the cardiac rate increases.

**The Mendelsohn-Graupner** method measures the length of time required for the heart rate to return to normal after exercise. The pulse is first taken when at rest. Various types of exercise (*e.g.*, raising dumbbells, stair climbing, ergostat, stationary bicycle) are then performed. Normally, the pulse rate should return to normal after a fair amount of work individualized according to the muscular make-up and physique of the person tested. The

assumption is that the longer the time required for the pulse rate to reach the normal, the less is the functional capacity of the heart.

**The Herz Muscle-contracting Test.**—If an individual slowly contracts a set of muscles (*e.g.*, those of the forearm) so that both extensor and flexor groups are employed, the author claims that the pulse rate remains the same or is slightly increased in healthy hearts, whereas in decompensated hearts, the rate is slowed.

**Graupner Method.**—With a measured amount of work, a normal heart reacts by an increase of blood pressure after mild exercise; a functionally weakened heart, by a fall of blood pressure.

**Plesch's** method depends upon a complicated compilation and deduction of the systolic output of the heart by the varying amounts of oxygen found in the arterial and venous systems.

Several methods attempt to study the cardiac function by taking the pulse rate in the sitting, standing, and recumbent positions, and observing the length of time required for the pulse to return to the normal.

**General Objections to Functional Efficiency Tests.**—It appears to me that there are several objections to the various methods of using blood pressure readings for tests of cardiac efficiency. Blood pressure depends upon important factors other than systolic output (Chapter XXV); the cardiac rate in circulatory failure often increases disproportionately to pulse pressure; increased cardiac work does not necessarily imply increased systolic output or increased blood pressure; the state of venous distention in the splanchnic area—an unknown factor because it cannot be measured—may considerably influence and vitiate the calculation. It is very difficult to eliminate or estimate the psychic factor. Finally, the various estimations can give but temporary information at the time the tests are made. Far simpler, more readily applicable, and clinically as accurate as the above tests, is the usual method of observing the patient during various forms of exercise—standing, walking, bending, etc.—and of estimating the cardiac reserve by noting the rapidity of pulse and respiration, dyspnœa and such subjective sensations as discomfort and exhaustion. Information derived from the patient's subjective sensations is of special importance, and cannot of course be calculated by any instrumental method. The above routine can be further amplified in appropriate cases by allowing the patient to follow his ordinary occupation, if not too laborious, for part of or the entire day, and then noting the effect upon his circulation and cardiac reserve. Such data, in addition to a very careful clinical examination, are, I believe, preferable to mathematical "efficiency tests" in the great majority of cases.

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## CHAPTER XV

## RHEUMATIC AND BACTERIAL ENDOCARDITIS—PREVALENCE, CLINICAL PHENOMENA, SYMPTOMATOLOGY, PROGNOSIS, THERAPY—DYNAMIC CONSIDERATIONS

**Prevalence of Heart Disease.**—There is undoubtedly a large proportion of the population affected with chronic vascular disease. If to this there be added those who suffer from correlated cardiosclerosis (including cardio-renal, cardio-vascular, hypertensive diseases, arteriosclerosis, "senile" heart changes), the sum must certainly be very large. Unless and until statistics

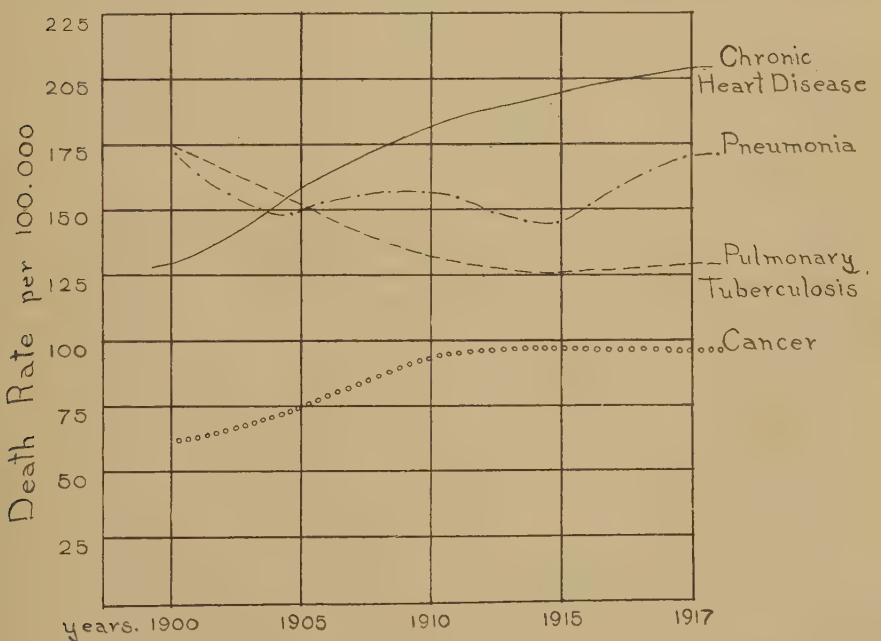


FIG. 275.—Curves showing comparative death rates from chronic heart disease, pulmonary, tuberculosis, pneumonia and cancer in 10 registration states.

more carefully individualize the type of heart disease that causes death, they can give only a general idea of the incidence of heart disease. Even thus, comparison with other frequent causes of death—pulmonary tuberculosis, cancer and pneumonia—shows that death from heart disease is commoner than from any of these (Fig. 275). Perhaps it is exactly our increasing knowledge of the correlation of kidney and vascular changes with myocardial

disease, and their report as "heart disease," that has caused this apparent increase during the last decade or so (see chart). During the draft examinations for the Great War a correlation of statistics showed an incidence of heart disease of 2 per cent. among those called for service. This seems to me higher than the actual facts warrant. It must be remembered, for example, that the medical examiners were themselves ill-prepared at the beginning and that many functional conditions, particularly functional murmurs and irregularities, were regarded as organic changes. Another evidence of unpreparedness for proper cardiac examination was the marked differences in ratio in rejected candidates among the various local boards. From my own experience in a local board where I examined several hundred men, I found very few who required rejection for actual heart disease. I have no statistics at hand, but the percentage of rejection was certainly not nearly 2 per cent. I therefore feel that an assumed incidence of organic heart disease of one in fifty young men called by draft boards probably overrates the actual prevalence of heart disease in younger individuals.

The etiology and pathology of heart disease have already been described (Chapters IV, V). For clinical purposes, I shall divide valvular endocarditis into rheumatic, bacterial, and chronic streptococcus viridans. Cardiac syphliis and cardiosclerosis are discussed separately.

## RHEUMATIC ENDOCARDITIS

Clinical phenomena accompanying acute endocarditis affecting any valve may be divided into the general rheumatic and the endocarditic manifestations. The former include the well known joint and muscular symptoms: Chorea, tonsillitis, erythema nodosum, subcutaneous fibroid nodules, etc. These rheumatic manifestations, however, do not always run parallel with the degree of endocardial involvement: The rheumatism may be mild, the endocarditis severe, or the reverse. I have, for example, noted the appearance of infections of the mitral valve following very mild catarrhal invasions of the upper air passages. One instance was that of a young, vigorous adult of twenty, suffering from mild pharyngeal catarrh. His highest temperature was 100.4°. After three days, a typical mitral lesion developed. Another instance is that of a young physician, age 32, who had recently passed satisfactory army tests with reference to his heart. A murmur was never present until it was accidently discovered some months later while being examined for life insurance. He never had rheumatism. Some weeks prior to the life insurance examination, he had pharyngitis lasting a few days. When I examined him, I found a loud systolic murmur heard over the mitral area and in the left axilla, typical of a mitral regurgitant lesion. There was no cardiac enlargement nor cardiac symptoms. I have the impression that such loud murmurs are not indicative of severe pathological damage to the valve but

are caused by mild valvulitis with dilatation of the mitral ring; probably it is the latter element which causes the loudness of the murmur in these cases.

Endocarditic and myocardial manifestations depend upon physical signs, and upon the type, course and complications of the valvular infection. The physical signs of the various lesions have been described (Chapter XIII). Upon their discovery rests not only the diagnosis of valvulitis but also the possibility of damage to the myocardium. That this possibility is present in every case of acute or recurrent endocarditis has already been indicated. Frank decompensation from severe involvement of the myocardium is extremely rare in acute endocarditis. There are, however, several signs and symptoms which, when present, tend to confirm the suspicion of myocarditis. Muffled indistinct heart sounds (not due to a pericardial effusion) in which the first sound resembles the second (so-called embryocardia), is an important sign of muscle involvement. Slight dyspnœa not accounted for by fever, tachycardia or exhaustion, is another. Continued rapid heart action and occasional extrasystoles after the fever and endocarditis have run their course are added hints of possible muscle disease. An important sign of myocarditis, only discoverable by electrocardiograms or polygrams, is the presence of prolonged conduction time from auricle to ventricle—partial heart block (Chapter X).

Writers have classified endocarditis according to special features (fever, cerebral complications, etc.), the assumed pathological process (verrucous, warty and papillary endocarditis) or upon the results of blood culture. Since the various groups merge, and since bacterial invasions (*e.g.*, streptococcus viridans) may complicate rheumatic endocarditis as a superadded bacteremia, sharp divisions are arbitrary and cannot be maintained. I propose therefore to describe the usual clinical type of simple rheumatic endocarditis, with such variations as are important.

The one prominent sign of acute rheumatic endocarditis is fever. The course of the fever as such is not characteristic. Its duration likewise follows no general type. Depending upon the severity of the endocarditic infection, fever may last from days to weeks. When the general rheumatic manifestations are still active, it may be impossible to trace the source of the fever, for it may then be due to rheumatism or endocarditis. When the rheumatic course becomes afebrile, and joint and muscular inflammation have ceased, any rise of temperature, especially if sustained, is extremely suggestive of an endocardial involvement. In other words, fever continuing after recession of rheumatic signs in the joints and elsewhere, is strongly indicative of endocarditis. Louder murmurs and cardiac irregularities strengthen this suspicion. Fever may be so slight that the temperature may have to be taken frequently in order to establish a febrile rise. This is especially true in the mild type of endocarditis of adults. More often there is a rise of temperature of one or two degrees; its usual accompaniments—lassitude, anorexia and headache—are then present. Hyperpyrexia, due to endocarditis alone

is uncommon. An initial chill or rigor is rare. It occasionally happens that there is no rise of temperature during the entire course of the endocarditis; this of course does not preclude the possibility of fever before the patient came under observation.

Certain prodromal symptoms following mild rheumatism or tonsillitis are suggestive of developing acute or recurring endocarditis, even in the absence of fever; this statement is particularly true in children. Such children, otherwise robust and healthy, complain of feeling tired and become anemic and less active. Epistaxis is by no means infrequent. These manifestations have no direct references to the heart, for the children are not especially dyspnœic, and physical signs of endocarditis are not then present. It is only when mild rheumatic symptoms occur, such as fleeting pains in the limbs, that a slight rise of temperature for a few hours may be observed; with it, the physical signs of endocarditis often become evident for the first time.

Between the very mild, almost non-febrile cases, and the commoner ones with moderate temperature, there exists all grades and types of febrile disturbances.

The physical signs of acute endocarditis are inconstant; they usually vary with the intensity of the process. The latter may be slow and gradual so that only after a long time do frank physical signs of valvulitis appear. On the other hand, physical signs, especially in children, may be immediate and unmistakable. The difficulty of interpretation of apical murmurs, especially in the presence of fever, has also been alluded to (Chapter XIII). Once established, the ausculatory phenomena of the valvular affections are those already described (Chapter XIII).

**Other Symptoms and Signs of Acute and Recurrent Rheumatic Endocarditis.**—The first hint of endocardial damage may be slight acceleration of the pulse. In adults the rate may reach 100; in children, 120 per minute. This pulse acceleration is commoner in children than in adults. It may precede the physical signs of endocarditis by several days. This occurred, for example, in a young woman of twenty-five with articular rheumatism and with no temperature at the time she came under observation. She suddenly developed tachycardia; it was only several days after rheumatic manifestations had ceased that the physical signs of endocarditis first presented themselves. Next in frequency to moderate pulse acceleration are extrasystoles. Paroxysmal tachycardia, auricular fibrillation and flutter are uncommon. Prolonged conduction time is sometimes found; this is an evidence of myocardial rather than of endocardial involvement. In very rare instances myocardial involvement shows itself by atrio-ventricular heart block (Chapter XI). New or disappearing patches of localized dry pericarditis, usually at the apex or over the left base, are occasional physical evidences, and offer clinical hints of the probability of acute endocarditis.

Subjective symptoms of various kinds are sometimes referred to the precordium during the course of acute endocarditis. They consist of indefinite

sensations of weight or pressure on the chest. Occasionally "thumping" sensations are complained of, although the heart rate is normal; this seems due to abnormally strong ventricular contractions. There are at times "sticking" or "stitch-like" precordial pains (Chapter XXIII). Pain may be present not only over the precordium, but may radiate to the left shoulder and neck, and to the left intercostal spaces. In general, the pains are not severe; they are more common when tachycardia is present.

Cardiac decompensation in any of its phases is very rare in simple acute rheumatic endocarditis.

**To Summarize.**—Fever, anemia, arrhythmias, or precordial sensations constitute suggestive signs of the onset of acute rheumatic endocarditis. Definite physical signs of a valvular lesion are necessary in order to clinch the diagnosis.

**Exacerbations and recurrences of simple rheumatic endocarditis** are, in general, marked by signs and symptoms similar to the original onset of the disease. Rheumatism, pronounced or obscure, is often present. In some respects however, rheumatic endocarditic recrudesence may differ from the primary attack. For example, pulse irregularities are more frequent. Tachycardia—simple pulse acceleration, or, more rarely, the paroxysmal type—is particularly common. Extrasystoles occur next in frequency. Auricular fibrillation, present throughout the entire exacerbation or coming in attacks, is by no means rare; in this respect, especially, exacerbations differ from the original onset. Auricular flutter is occasionally observed. Subjective precordial sensations are more common; they vary from sensations of pressure to frank attacks of precordial distress. The physical signs of the valvular lesions are more pronounced unless they are masked by rapid or irregular heart action. Mild disturbances of compensation—slight edema of the legs, enlarged liver, dyspnœa, etc.—begin to make their appearance.

**Chronic Rheumatic Endocarditis.**—Depending on the duration and frequency of rheumatic exacerbations, the symptoms and signs of recurring endocarditis merge into those of chronic endocarditis, embracing as it does the great majority of cases of valvular heart disease. Patients with chronic endocarditis and perfect compensation may continue in good health for years; cardiac symptoms may never occur. These are the fortunate instances in which the disease has become quiescent, and the cardiac damage, especially to the musculature, has not been extreme. The chief symptoms of chronic rheumatic endocarditis are due to myocardial insufficiency and to decompensation. They have already been described in detail. It remains now to differentiate the special signs and symptoms constituting the syndromes of decompensation in chronic rheumatic valvular lesions, as well as some considerations regarding the special dynamics involved.

**Decompensated Mitral Regurgitation (Mitral Incompetency). Dynamic Considerations.**—When the mitral valve is rendered incompetent in a laboratory model of the circulation, there is a rise of auricular and a fall of

arterial pressure; the venous system is over-filled and there is a decreased transfer of blood from the venous to the arterial side. With the "heart" (pump) rate unaltered, there is a decreased ventricular output and a fall of arterial pressure. The "pulse" becomes smaller in amplitude. In animal experiments, MacCallum and McClure have shown similar results: In the experimentally produced mitral leak, there is a fall of pressure in the systemic arteries and a rise of pressure in the left auricle. The pressure in the pulmonary artery may rise or fall, depending on the degree of mitral insufficiency produced. When the insufficiency is extreme, the pressure in the pulmonary artery falls; when slight, the pressure usually rises. In either case, there is pulmonary congestion. But important as these laboratory and experimental observations are in support of the older "back pressure" theory (in this instance, failure of the right heart) in relation to experimental valvular leaks and their consequences, they are not entirely comparable to what takes place in the human being. In animals and in laboratory models, the leak is a sudden operation and hence requires sudden accommodation on the part of the cardiovascular apparatus. In the human being, the pathological process is usually a slow and gradual one, so that the heart, and the systemic and pulmonary circuits can slowly accommodate themselves to changes of pressure and of blood output. It is none the less true that pulmonaty signs often represent the initial changes in decompensated mitral regurgitant lesions (see below), hence it seems probable that at least in some cases, "back pressure" plays a clinical role. It should be remarked that a small regular pulse (thatis, one of small amplitude), often present in mitral regurgitation and regarded by some as significant of decompensation, does not necessarily imply a correspondingly lowered blood pressure, as can be readily shown by clinical blood pressure observation (Chapter XXV).

**Clinical Syndrome of Mitral Regurgitation.**—With beginning heart failure, venous stasis affecting the pulmonary circuit is often an early symptom; hence bronchitis is one of the first, sometimes the only early manifestation. Bronchitis may range from a short, hacking, unproductive cough to one with numerous soft, mucous rales and areas of sibilant breathing scattered over both lungs. This complex with its physical signs accurately resembles true bronchial asthma, from which it must be differentiated. The signs of endocarditis and heart failure are usually sufficient to make the differentiation. Hemoptysis or expectoration of blood mixed with muco-pus is not infrequent. Patients with mitral regurgitation and heart failure are as a rule cyanotic. When cyanosis is extreme, the lips and extremities are bluish; the conjunctivæ, suffused and discolored. Even with marked cyanosis, dyspnœa may be mild when the patient is resting quietly. Severe decompensation is accompanied by the visceral congestion and dependent edema already described (Chapter XIV). The usual subjective phenomenon is a feeling of weight or pressure on the chest; severe precordial pains are rare. On the other hand, sensitiveness to pressure in the epigastrium is common with or

without gastric phenomena. Somnolence with a fair output of urine in the extremely decompensated is probably due to cerebral congestion and edema (Chapter XIV).

Moderate pulse acceleration is the rule, extreme pulse acceleration or paroxysmal tachycardia is exceptional. The usual cardiac irregularities are extrasystoles (commonly ventricular) and auricular fibrillation; extrasystoles are observed in the beginning, auricular fibrillation in the later stages of decompensation.

**Decompensated Mitral Stenosis. Dynamic Considerations.**—In the circulatory model, mitral stenosis causes a rise of venous, and a fall of arterial pressure, with lessening of the amplitude of the pulse wave. Narrowing of the mitral orifice can be produced experimentally upon animals by placing a ligature around the auriculo-ventricular groove, or by placing a small inflatable balloon within the left auricle. There follows a rise of pressure in the left auricle and pulmonary artery, and a fall in the systemic arterial pressure. It is commonly assumed that similar conditions occur in the human being: Namely, a rise of pressure in the pulmonary circuit; as a consequence, increased pressure in the right ventricle occurs, finally leading to dilatation of the tricuspid ring and functional regurgitation in that orifice. A murmur due to functional tricuspid regurgitation is not infrequent in clinical cases of decompensated mitral stenosis. (See Chapter XIII.) Besides purely mechanical considerations of pressure differences, there are probably important pathological factors involved (myocarditis) in the production of this type of tricuspid regurgitation in the human being.

**Clinical Syndrome of Decompensated Mitral Stenosis.**—A subjective feeling of palpitation combined with slight tachycardia is one of the earliest symptoms. This is not due to moderate pulse acceleration alone, but also to increased violence of the heart action. Slight dyspnœa accompanies the palpitation. Cyanosis is a comparatively late symptom and marks the period of severe decompensation. An enlarged and distinctly pulsating liver may be present even when cardiac failure is not extreme. Sharp, precordial "sticking" pains are common; they usually accompany the tachycardia and are present without pericarditis. Paralysis of the vocal cord due to paralysis of the left recurrent laryngeal nerve occasionally occurs.

Ortner in 1897 first described the connection between mitral stenosis and paralysis of the left recurrent nerve. Since then, reports of thirty-two cases have been collected from the literature by Cuisset up to 1912. The theories held accountable for the paralysis may be recapitulated as follows:

**I. Ortner's Theory.**—Recurrent paralysis is due to pressure of the enlarged left auricle upon the nerve.

**II. Kraus' Theory.**—The right ventricular dilatation present in mitral stenosis causes displacement of the heart to the right, with consequent dragging on the aortic ligament and arch, and resultant stretching and paralysis of the nerve.

**III. Alexander's Theory.**—The pulmonary artery, either by its own enlargement or indirectly by enlargement of the left auricle, is pressed against the nerve and aortic arch.

IV. The nerve may be compressed and caught between bands of pericardial and mediastinal adhesions.

All these theories have had some necropsy support. Normally, the pulmonary artery is situated under, and divides immediately beneath, the aortic arch; below the pulmonary artery is the left auricle. It therefore seems improbable that the left auricle, even when extremely enlarged, can exert sufficient direct pressure to produce recurrent paralysis, unless the auricle is jammed between, or is adherent to, the pulmonary artery and aorta. This was the finding in one case which came to necropsy. In another case, the left auricle was the size of a small fist and was found pressing against the nerve. From careful anatomic studies of frozen sections, Fetterolf and Norris state that the effect of left auricular dilatation is pressure of the left pulmonary artery against the aorta, and of the left pulmonary vein against the pulmonary artery, thus forcing the latter against the aorta. They believe that direct pressure alone of the enlarged left auricle can scarcely produce paralysis unless the auricle is squeezed between or is adherent to the pulmonary artery and aorta. They conclude that anything which will dilate or force upward the left auricle, the left pulmonary vein, or left pulmonary artery will tend to produce paralysis, and that the latter must finally be caused by the nerve being "squeezed between the left pulmonary artery and aorta or aortic ligament." This conclusion bears out the common knowledge of the normal close juxtaposition of pulmonary artery and aorta (Chapter I). A similar conclusion was reached by Frischauer, who, in a case at necropsy, found the nerve compressed between the left pulmonary artery and aorta by the pressure of the dilated left auricle and pulmonary vein.

I had an opportunity to observe a patient with mitral stenosis in whom there was not only paralysis of the left recurrent nerve, but also a marked difference in the radial pulse. The history and the result of the examination in this case are as follows:

Female, aged 25, married two years, no children or pregnancies, had a severe attack of inflammatory rheumatism thirteen years ago. There were no cardiac symptoms until three years ago; at that time palpitation began. During the last year, she had also become dyspnœic. Hoarseness commenced six months ago. Examination of the larynx showed that, owing to left recurrent paralysis, the left vocal cord was immovable and somewhat shortened, and in the cardaveric position. The voice was indistinct and hoarse. The examination of the cardio-vascular system revealed the following: There was a noticeable difference between the right and left radial arteries on palpation, particularly when the arms were extended above the head. The systolic and diastolic blood pressure of the right brachial were respectively 110 and 80 m.m. of mercury; of the left, 82 and 70 m.m. There was no perceptible dif-

ference in the carotid pulsations. The pulse was regular, the rate, 100 per minute. There was vigorous visible precordial pulsation; a diastolic thrill was felt, and a loud rumbling, presystolic murmur was heard at the apex. The aortic sounds were normal. Over the pulmonary area there was a somewhat rough systolic murmur and an exceedingly accentuated snappy second sound; the sharp click of the pulmonary valve closure was also evident on palpation. A dry pericardial friction rub was heard over the lower sternum; three weeks later, there was evidence of fresh pericarditis over the pulmonary area. There was no systolic apical retraction. Orthodiascopic

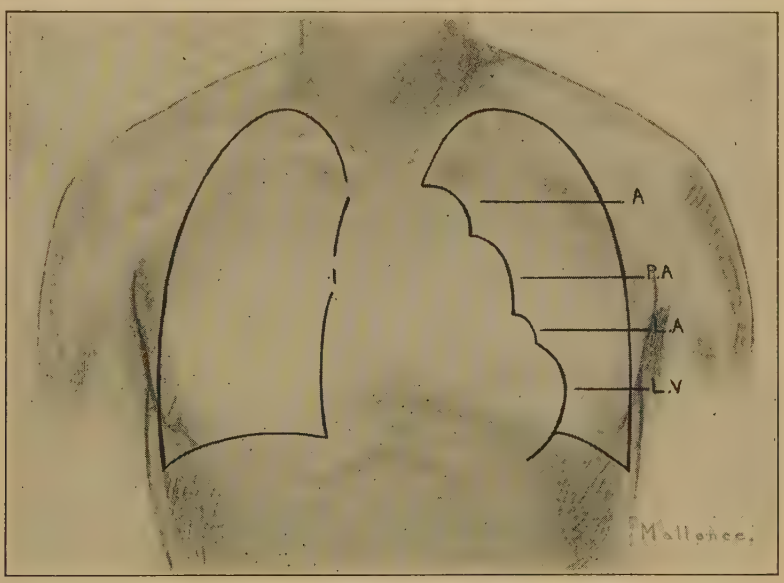


FIG. 276.—Orthodiascopic tracing of a case of mitral stenosis with paralysis of the left recurrent nerve.
A, aortic outline; P.A., enlarged pulmonary artery; L.A., left auricle; L.V., left ventricle.

examination (Fig. 276) revealed a short aortic bulge (A). The outline of the aorta was apparently encroached upon by the very much dilated pulmonary artery (P.A.). The left auricle (L.A.), though not enlarged in the tracing, was seen in the fluoroscope to overlap the pulmonary artery to some extent. The left ventricle (L.V.) and the right side of the heart were also somewhat enlarged. The electrocardiogram showed a negative S III, and a notched and enlarged P in the first lead.

In this case, the fluoroscope showed no evidence of abnormal right ventricular enlargement; the heart was dilated chiefly to the left. This evidence of left ventricular enlargement tends to discredit Kraus' theory as to the etiology of recurrent paralysis in mitral stenosis. The orthodiascopic findings and the physical signs over the pulmonary artery in my case leads to the conclusion that the recurrent nerve paralysis was due to dilatation and

pressure of the pulmonary artery against the aorta; whether or not the left auricle was a factor in pushing up the pulmonary artery, it is impossible to state. Lian and Marcorelles insist upon the presence of old thrombosis of the left auricle, or of a mediastinitis as contributory causes. Pericarditis was present in my patient; it was at first confined to the precordial area over the lower sternum; later it involved the area over the pulmonary artery. This may have been an added factor in binding down the dilated pulmonary artery.

A unique feature of the case was the marked difference in the right and left radial arteries, and in brachial blood pressures. This, in conjunction with paralysis of the vocal cord, formed a striking resemblance to the clinical syndrome of aortic aneurism. The difference in brachial pressure seemed due to the dilated pulmonary artery being wedged under the aortic arch opposite the origin of the left subclavian artery, with a pressure sufficient to produce interference with the circulation of the subclavian, and, consequently, of the left brachial.

Attacks of paroxysmal tachycardia are comparatively frequent in decompensated mitral stenosis. Emboli produce occasional complications. Hemiplegia and aphasia may follow cerebral emboli. As an instance, one of my patients, a woman aged 50 with a mitral stenotic lesion, had aphasia for ten years from a cerebral embolus. A large artery of the extremity may be plugged by an embolus and cause gangrene. This happened in a patient of forty with mitral stenosis and finally led to amputation of the leg; the patient recovered. Hemoptyses from pulmonary emboli are not uncommon. Such cases must be carefully distinguished from the hemorrhage of pulmonary tuberculosis. The physical signs of mitral stenosis, and the site and nature of the pulmonary signs usually make the differentiation easy. Emboli do not necessarily occur with symptoms of recurring endocarditis, nor can they be regarded as evidence of cardiac failure in the true sense. Indeed, they usually antedate severe dyspnœa, edema and visceral congestion by months or years. These embolic "accidents" seem in the main due to mechanical breaking off of vegetations on the mitral valves from overforceful and abnormally rapid heart action. Another frequent characteristic of mitral stenosis, already described (Chapter XVIII), is its susceptibility to nervous influences of various sorts. It is probably this which causes disturbances of heart action from various sources. Indeed, it is apparently this nerve lability which causes various referred disturbances of the gastro-intestinal canal. The gastric syndrome is that usually associated with hyperacidity or even of gastric ulcer. It is therefore extremely important to study such gastric phenomena from the cardiac aspect, otherwise patients are unnecessarily rigidly dieted and put upon ulcer treatment.

Severe decompensation in mitral stenosis usually occurs with the onset of auricular fibrillation, with the signs and symptoms already described. The arrhythmia, once established, often remains permanently. Occasion-

ally, however, fibrillation occurs with each fresh exacerbation of endocarditis. Auricular fibrillation is found much oftener in mitral stenosis than in any other valvular disease.

**Aortic Stenosis—Dynamic Considerations.**—In the circulatory model, aortic stenosis causes a marked increase of intra-ventricular pressure during systole, and a fall of arterial pressure during both systole and diastole. The "pulse" wave shows a slow ascent and a rounded top (*pulsus tardus*, Chapter VII). Similar observations have been made upon the experimental animal; when a marked degree of aortic stenosis is produced, the rise of intraventricular pressure is correspondingly marked. The time of systole is prolonged, however, so that the amount of blood ejected into the circulation is not much decreased.

**Clinical Syndrome of Heart Failure in Aortic Stenosis.**—As already mentioned, aortic stenosis as an isolated valvular lesion is rare. The symptoms of heart failure depend chiefly upon the degree of ventricular hypertrophy with its implied lowered cardiac efficiency. Marked decompensation is a late symptom, and is found only when hypertrophy becomes extreme. Congestion and auricular fibrillation occur only in the terminal stages. Among earlier symptoms are those due to cerebral anemia (dizziness and faintness). Other beginning manifestations consist of a subjective feeling of palpitation and of moderate tachycardia. Embolic infarcts in the viscera or extremities are occasional complications.

**Decompensated Aortic Regurgitation—Dynamic Considerations.**—In the laboratory model the production of aortic regurgitation is followed by a fall of the diastolic pressure in the arterial system; there is no constant change in systolic blood pressure. Similar results have been obtained in animal experimentation. The cause of the fall of diastolic pressure, however, is still in dispute. According to the older assumption, the diastolic fall of pressure is massive back flow of blood during ventricular diastole. More recent observations (Stewart, Wiggers) demonstrate that the amount of back flow is after all but a small fraction of the amount ejected during systole. This was shown by cardiometer experiments and by the fact that the rate of ventricular relaxation (Chapter III) remained unaltered. Stewart attributed the diastolic fall of arterial pressure to reflex dilatation of the peripheral vessels. As the result of studies of subclavian and intraventricular pressure curves, Wiggers maintains that peripheral dilatation is not a factor in this diastolic fall but that it must be attributed to a "back leak of pressure into the ventricle with or without an appreciable back flow of blood."

The clinical application of these facts seems to be that the amount of blood regurgitating into the ventricle is probably very slight and that "back pressure" becomes a factor only when for any reason the muscular efficiency of the left ventricle becomes subnormal.

**Clinical Syndromes of Decompensated Aortic Regurgitation.**—The patients are characteristically prone to suffer from attacks of tachycardia

(often paroxysmal), and from very severe precordial pains. Pains, dyspnœa and tachycardia frequently occur in nocturnal attacks. Mechanical interference with the coronary flow by the aortic lesion may be a cause of some of these attacks. The pains are usually severe, sometimes agonizing (q. v. Cardio-vascular Clinics). Their favorite site is the precordium; they may radiate to the arms, neck, abdomen, and even to the legs. Fright, excitement, over-exertion, or nervous strain may be sufficient to initiate an attack of pain or of tachycardia, especially when ventricular hypertrophy is far advanced. Such attacks may also represent the only evidence of fresh endocarditis. Susceptibility of the heart to nerve influences of the most varied kinds, a condition common in aortic regurgitation, depends chiefly, I believe, upon continued mechanical "insults" from aortic hyperactivity to branches of the cardiac plexus surrounding the root of the aorta (Chapters I, XVI). This may well produce an altered state of nerve tone and of nerve control, leaving the heart readily susceptible to extraneous influences.

Hemoptyses occasionally occur with the tachycardial attacks; they seem due to acute pulmonary congestion from rapid heart action. More rarely, hemoptysis may be the result of a pulmonary infarct. Another occasional complication of aortic regurgitation consists in periods of somnolence merging into stupor. In one patient, a girl of 17 with a typical rheumatic regurgitant lesion and tremendous left ventricular hypertrophy, these cerebral manifestations were usually ushered in by fever up to 103°, and by paroxysms of auricular fibrillation. Cyanosis was not present. The dyspnœa during these attacks was not more marked than usual. The fever and fibrillation seemed due to recrudescence of endocarditis. Meningitis could be excluded. The accompanying somnolence, and later, stupor, in this and other attacks, could scarcely be accounted for by the fever alone or upon the supposition of a toxemia, for the patient had had a severe and long continued pneumonia with higher fever, without any cerebral manifestations. In patients with well marked aortic regurgitation, cerebral anemia from disturbed circulation in the brain is perhaps the underlying cause for such attacks of somnolence and semi-stupor.

Visceral congestion, edema, and permanent auricular fibrillation represent late stages of decompensation in aortic regurgitation.

Decompensation with rheumatic disease of the pulmonary and tricuspid valves is not described because rheumatic endocarditis affecting these valves is very rare, especially as isolated lesions.

**Therapy of Rheumatic Endocarditis without Decompensation.**—During the acute stages, aside from the specific action of drugs of the salicylate group, we possess no medication that can influence the pathological process affecting the endocardium and valvular structures. Therefore in acute rheumatic endocarditis, or in endocarditic exacerbations accompanied by general rheumatic manifestations, the salicylates, preferably salicylate of soda, are indicated. Clinical observation has shown that the salicylates often control

rheumatic manifestations; it is therefore assumed that they may control the rheumatic poison affecting the heart. I prefer to give the salicylate of soda in 15 grain doses hourly until tinnitus occurs, or until six doses have been taken; thereafter, the same dose should be given three times daily. The futility of sera and vaccines is discussed later (Chapter XX). When fever is present the diet should be bland, mainly fluid. As fever decreases and the patient's appetite returns, semi-solid and finally ordinary diet may be permitted. I find no special value in the restriction of the amount or kind of meat when the gastro-intestinal and kidney functions are normal and when the patient craves food (Chapter XX). Rest in bed should be enjoined during the period of the acute endocarditic attack. The plan, sometimes followed, of keeping patients in bed many weeks or even months after the acute stage has passed, possesses no value in aiding the circulation or in preventing reinfection (Chapter XXII). From three to five weeks I consider the average time for rest in bed after the acute manifestations have passed. In those with rapid heart action or with precordial discomfort, an ice bag should be placed over the heart; it should be kept in a proper sling so that the patient is not continually annoyed by its slipping. The length of time that ice is to be applied depends chiefly upon the patient's feelings. I have occasionally found more relief from hot applications (hot water bag, electric pad) than from cold. If tachycardia is marked, or if patients are restive, luminal in one and one half grain doses, the bromides alone in fifteen grain doses, or bromides combined with small doses of morphine, are indicated. Occasionally such hypnotics as chloral and veronal are required. Digitalis is rarely indicated, for decompensation does not often occur in acute endocarditis. An exception may be the presence of tachycardia with acute dilatation and dyspnœa, a condition more frequent in children than in adults.

**In chronic rheumatic endocarditis,** when no decompensation exists, drug medication intended to affect the heart and circulation is not indicated. Here, questions regarding proper exercise, vocation and occupation, play important roles (Chapter XXII). The therapeusis of decompensation in chronic endocarditis is described, to a great extent, in another chapter (Chapter XIV). Although medication is in general the same for the various valvular lesions it should again be emphasized that the same therapy is not followed by uniform results. The most beneficial results are derived from digitalis alone or in conjunction with quinidin sulphate (Chapter XX) when employed in decompensated mitral stenotic lesions with auricular fibrillation. In the heart failure of aortic disease with marked hypertrophy, digitalis has little or no effect in relieving decompensation, even in the presence of auricular fibrillation. The influence of foci of infection—teeth, tonsils, etc.—is elsewhere described (Chapter XXII).

## PROGNOSIS IN RHEUMATIC ENDOCARDITIS

It is naturally impossible to give categoric rules which will infallibly guide one in the prognosis. It is always necessary to study the disease in all its various aspects, as well as how it affects the individual patient. In chronic cases, the following general statements may be permitted, namely; prognosis depends more upon the condition of the cardiac musculature than upon the valve that is affected; and the greater the amount of hypertrophy, the worse the prognosis, especially as regards longevity.

For clinical purposes the question of prognosis in rheumatic endocarditis may be divided into the *acute* and *subacute*, the *quiescent* and the *chronic* stages. The first two are here grouped together because they overlap so frequently that a sharp distinction is impossible.

In the *acute* and *subacute* stages, the main prognostic problem is to determine, if possible, the virulence of the infection. Although, as before stated (Chapters IV, V), a few observers claim to have isolated the specific organism from the blood of patients suffering from rheumatic endocarditis, this has not been substantiated by other bacteriologists; hence blood examination for bacteria cannot at present be used to gauge the severity of the infection. In addition to information gained from physical signs, most reliance regarding the degree of virulence in rheumatic valvular infections must be placed upon such clinical manifestations as the acuteness and severity of the onset, the frequency of chills, the presence of hyperpyrexia, vomiting, leucocytosis, severe anemia and delirium, and upon such complications as pericarditis and pleurisy with effusion. If both the aortic and mitral valves are affected, the acute and subacute stages are apt to last a longer time than when one valve alone is diseased, and the prognosis is correspondingly worse. Patients with aortic lesions are somewhat more prone to complications arising from the virulence of the infection than are those with mitral lesions. During the time that systemic rheumatic manifestations are present, the endocarditis cannot be regarded as quiescent, even if there be no clinical manifestations of recrudescence. However, more direct evidence of fresh endocarditis may be found in increased intensity and harshness of endocardial murmurs, temporary arrhythmias, precordial "sticking" pains, and, occasionally, in embolic infarcts in the brain, lungs or kidneys. Slight rises of temperature, if not otherwise accounted for, are to be regarded as probable signs of the continuation of the endocarditic process. Another highly suggestive evidence is anemia, severe or moderate, not responding to the usual treatment.

Occasionally the infection is mild, fever and other inflammatory manifestations cease, the valvular lesion quickly becomes quiescent, and indeed soon eludes all signs of ever having been present. Such cases usually occur in children and young adults, and apparently represent instances in which healing begins soon after the mild endocardial infection has run its course. The existence of such cases has been corroborated by necropsy findings.

If the physical signs and clinical phenomena indicate an active endocarditis lasting for a month or longer, the outlook for a "quiescent" period, and therefore for a favorable prognosis, becomes correspondingly poor. These patients may die of embolic infarcts, or of superadded bacteremias, such as pneumococcemia or streptococcus viridans infections. Decompensation usually plays a minor role.

**Quiescent Stage.**—In the fortunate cases, this stage represents uninterrupted convalescence from the acute and subacute, with complete cessation of active signs of endocarditis. These patients with their recurrences and exacerbations comprise the great class of chronic rheumatic heart cases. Conceiving the quiescent stage as convalescence from the acute, decompensation is rare, while the chances of the lesion becoming "chronic" and remaining quiescent are good. The prognosis then depends upon that of chronic endocarditis.

In *chronic quiescent* cases, the cardinal point in prognosis is the consideration, not of the number or varieties of murmurs present, but the extent and damage to the myocardium, factors usually decided by a study of the muscular and circulatory efficiency of the heart. If myocardial efficiency and cardiac reserve are good, a correspondingly good prognosis may be given. Regarding prognosis from the standpoint of longevity, the above observations must be combined with a general knowledge of the average duration of life in the various types of valvular lesions; but each case must nevertheless be studied individually in the attempt to gauge the probable span of life.

It has been demonstrated statistically that patients with mitral insufficiency live longest; individuals with this lesion living to ripe old age are not uncommonly encountered. The usual duration of life in mitral stenosis is much less; one of the reasons for this is that such cases are prone to develop auricular fibrillation, and with it, the usual train of symptoms and dangers arising from decompensation. In aortic regurgitation, massive ventricular hypertrophies (*cor bovinum*) are common; yet years may elapse until death occurs from decompensation, emboli or from some superadded acute infective process. In general, however, the chances for longevity with this lesion correspond somewhat to the degree of ventricular hypertrophy; where this is marked or extreme, patients are not apt to live beyond middle age.

**Acute Bacterial Endocarditis.**—Other terms for this infection are malignant, ulcerative, infectious and septic endocarditis. Bacterial endocarditis as an acute process engrafted upon a chronic valvular lesion is not here included. It has already been pointed out that, although not proven, rheumatic endocarditis is probably of bacterial origin. But acute bacterial endocarditis differs so markedly from the rheumatic type, and even from streptococcus viridans infection (itself a bacteremia), that it deserves individual description.

Acute bacterial endocarditis is almost always a secondary process; its presence as a primary lesion has indeed been denied by several writers.

It may complicate erysipelas, pneumonia, puerperal fever; in fact, any bacteremia. It is occasionally present as a terminal infection. Although the course of acute bacterial endocarditis is essentially acute, occasional cases of comparatively long duration have been described. In acute staphylococcic infections, the portal of entry is the skin or an osteomyelitic focus; in streptococcus pyogenis, the mucous membrane of the throat or uterus. Gonococci, also streptococci and staphylococci, may enter the general circulation from the genito-urinary tract; pneumococci, from the respiratory tract.

The clinical picture of acute bacterial endocarditis is essentially that of a more or less virulent bacteremia, with all its protean and manifold characteristics. The disease rarely lasts more than four weeks. There are often no cardiac manifestations; no murmurs, pain, dyspnœa or other signs of cardiac involvement.

The clinical manifestations are sometimes divided into the typhoid, septicemic, cerebral, etc. These varying characteristics depend upon the virulence of the invading organism, and upon the underlying cause of the bacteremia (erysipelas, puerperal septicemia, etc.).

The course of bacterial endocarditis is usually marked by high fever; daily chills are common; long remissions of temperature are rare. Sweating is often profuse. The onset of the endocarditis may be marked only by an intensification of the fever from the original source of infection, or by an initial rigor. Added hints of its onset may be found in sudden increased pulse rapidity, in sticking pains over the precordium, in the friction rub of dry pericarditis. When pericarditis is present, there is usually endocardial involvement as well.

Septic infarcts into various organs mark the stormy progress of the disease. Thus, cerebral infarcts may produce paralysis and unconsciousness, or, occasionally, purulent meningitis; symptoms from the latter may then dominate the clinical picture. Hematuria, sometimes abundant, may follow a renal infarct; pneumonia, an infarct in the lungs. Of frequent occurrence are petechiæ. They are usually numerous, especially upon the oral and conjunctival mucous membranes. The petechiæ sometimes contain pinpoint pustules in their centers. Occasionally, patchy erythematous areas in the skin, resembling urticaria, may be found. Various joints, especially the larger ones, may be involved in a purulent or sero-purulent inflammatory process.

The usual general manifestations of sepsis, or of a septic (typhoid) condition are common. The patients may be actively delirious, or be stuperous and comatose. There may be coma vigil with a dry and coated tongue.

As a rule the left heart alone is affected. It is, however, not unusual to have the right heart also affected, or even the right heart to the exclusion of the left.

Medication, including sera and vaccines, possess no value. The treatment is symptomatic only.

## SUBACUTE AND CHRONIC STREPTOCOCCUS VIRIDANS INFECTIONS

I have made no attempt to divide the disease into two distinct stages, for the terms "subacute" and "chronic" depend chiefly on differences of duration rather than of symptomatology. The usual duration is from a month to one year or more, hence either term may be appropriate in individual cases.

**Pathological Features.**—Of the bacterial infections of the heart, that of the streptococcus viridans has been most exhaustively studied. The process is almost always engrafted upon a chronic rheumatic infection. Occasionally, congenital lesions form the nidus. The pathological process consists of vegetative proliferative masses of grayish, greenish or pink color. Their main site is the mitral valve. Here they may form a few soft, friable masses, or the valve may be encrusted with large polypoid lesions. The latter may then extend along the left auricular wall above, and the chordæ tendinæ below. The process on the chordæ tendinæ occasionally leads to ulceration and rupture of these structures. Similar sequelæ are sometimes found as the result of vegetations on the mitral. Proliferative lesions on the aortic cusps and walls are less common and less extensive than on the mitral. Mycotic aneurisms of the valvés are occasionally found.

Characteristic changes in the kidney depend chiefly on the presence of infarcts. When pyogenic, they give rise to numerous small congested areas containing minute purulent foci; these are readily seen when the capsule is stripped from the kidney. Non-pyogenic bland infarcts occasionally occur; they show the changes usual to anemic necrosis; the infarcted areas are wedge-shaped and may be several centimeters in depth. When they are recent, the cut surface is yellow; the color becomes paler with the process of organization. Another type of infarct, assumed as pathognomonic of chronic streptococcus viridans, is embolic focal nephritis. Some characteristic changes are then found in the glomeruli; a part of, or an entire tuft may be involved. The capillaries are congested, the glomeruli contain a fibrinous exudate, the glomerular epithelium becomes swollen, and finally desquamates. The adjacent parietal layers of Bowman's capsule are often involved in the necrotic process, so that the entire necrotic area becomes semilunar in shape. The mass eventually organizes. There is a growth of epithelial cells from the healthy adjacent Bowman's capsules; this growth finally covers the lateral surface of the mass. When healing is completed, the result is a hyaline area of pyramidal shape.

The cerebral lesions consist chiefly of softening from emboli. Occasionally, cerebral hemorrhages from rupture of embolic aneurisms of the cerebral vessels have been observed.

**Clinical Features.**—The pathological features enumerated, and the fact that streptococcus viridans infection is a bacteremia, made it apparent that the clinical complex varies considerably. The predominant clinical

features depend on the toxemia, on focal emboli in the various organs and viscera, and on the pronounced pathological changes in the heart and kidneys.

The onset of the streptococcus viridans affection is usually insidious; the only complaint for days or weeks preceding the definite febrile period may be slight general malaise or mild anorexia. Sometimes the initial symptoms resemble those of other febrile invasions: rigors, chills, vomiting and fever between 101° and 103°. Occasionally, the onset is even more acute, and the daily temperature reaches 104° or 105°, with remissions resembling malaria. Such initial symptoms usually betoken a stormy and comparatively short course for the viridans infection. When the disease is once established, it is usual to have a febrile period from one to three weeks, with afebrile intervals of about the same duration. Each new invasion, at its beginning, may be marked by severe chills and hyperpyrexia; temperature up to 106° is occasionally encountered. The disease may thus continue for months until emboli or exhaustion produce death.

Petechiæ represent one of the most prominent and frequent manifestations. They are due to small capillary emboli. On the mucous membrane they appear as small, isolated red spots, with pale centers surrounded by an area of congestion, and sharply defined from the normal mucous membrane. The favorite site of petechiæ is the lower conjunctiva. Here they are characteristically found at the angulation of the vessels; the entire sac should be well exposed by pressing back the eyeball. Occasionally, petechiæ are found, not in the lower but in the upper lid. They are also found in various parts of the oral and even the pharyngeal mucous membranes. In one of my patients in whom petechiæ often appeared in crops on the skin as well as on the conjunctivæ and buccal mucous membrane, I found in addition a few in the anterior urethra.

Skin petechiæ resemble those of the mucous membrane. They are usually small and punctate. Occasionally, they become somewhat larger. Very rarely they are surmounted by minute pustules. While isolated petechiæ are the rule, it is not unusual to have them appear in crops over various parts of the body. Occasionally their appearance is attended by severe neuralgic pains in the area corresponding to the location of the rash. The cause of such pains is not apparent. The petechiæ are not tender; they usually disappear within one week. They sometimes leave small pigmented spots for a few days; these must be carefully distinguished from other pigmentations, especially freckles.

In addition to petechiæ, other skin manifestations are not uncommon in this infection. Painful erythematous nodules, slightly red and somewhat tender (Heubner's nodes) may occur in the terminal phalanges, especially of the fingers. The nodules, usually multiple, disappear within a few days; they are probably of embolic origin. Pyogenic emboli in the deeper dermal arteries may produce skin gangrene or necrosis. This occurred in one of my cases and finally resulted in sloughing of the entire scrotum. Emboli are

sometimes scattered, shower-like, through the smaller arteries of the extremities, producing numerous small areas of deep or superficial gangrene, and necrosis in fingers and toes. This was well illustrated in a woman aged 50 with streptococcus viridans and a decompensated double mitral lesion. She developed gangrenous areas on several digits and toes, and on the dorsum of both hands. She died one week later.

The hematological finding in streptococcus viridans infections is sometimes of great importance. While a mild secondary anemia with moderate leucocytosis and a high polynuclear count is the rule, anemia may become so marked as to overshadow the clinical picture, even leading to the erroneous diagnosis of non-organic, hemic instead of organic, endocardial murmurs. Occasionally, the blood picture and the hemoglobin percentage so closely resemble pernicious anemia that patients have been treated for the latter disease; only at necropsy has the correct diagnosis been established.

**Splenic enlargement** is quite common; it may vary from moderate increase in size to one reaching several inches below the free border of the ribs. At necropsy, infarcts of various sizes are almost regularly found in this organ.

**Changes in the Urine.**—A trace of albumin and a few casts are frequently found. More significant, when traced to the kidney as its source, is the presence of a microscopic amount of blood. Macroscopic hematuria is rarely present. The streptococcus viridans can only occasionally be cultured from a catheterized urine specimen. In exceptional instances renal signs and symptoms predominate the clinical picture, as in one of my patients. There was unilateral renal colic and bleeding. A blood culture taken at that time was sterile. A nephrectomy was done; the affected kidney was found twice the normal size and riddled with infarcts. The patient died two weeks later. Another blood culture, taken two days before death, showed the streptococcus viridans. At necropsy, valvular lesions characteristic of streptococcus viridans were found.

**Nervous manifestations** are frequent. They depend on one or several factors, all of which must be etiologically considered. Stupor, for example, may be due to toxemia, hyperpyrexia, meningitis, cerebral infarcts or to uremia. An instance of the difficulties with which the etiology of this one symptom may be surrounded is that of a woman aged 45, admitted to the hospital in semi-stupor. A double mitral lesion, auricular fibrillation, and irregular fever were present. The urine showed microscopic hematuria and a few renal elements. Two blood cultures were negative. A spinal tap was unsatisfactory. Semi-stupor finally merged into coma and death. Clinically, the symptoms seemed due to meningitis. At necropsy, a double mitral lesion, splenic infarcts and the glomerular kidney changes of streptococcus viridans were found. Meningitis was not present. Toxemia was the apparent cause of stupor and coma. The most common cerebral lesions in streptococcus viridans infections are hemiplegias; monoplegias are rare.

Occasionally pareses, not paralyses, occur, but disappear during the course of the disease. Meningitis is usually a terminal event; its clinical signs may be disguised by toxic symptoms.

Pneumonic complications are not often found in chronic streptococcus viridans infections. Enterorrhagia from an intestinal infarct is occasionally met with. Retinal hemorrhages sometimes occur.

Cardiac symptoms (precordial discomfort, decompensation, etc.) are not regularly present, and then but late in the infection. Even when valvular vegetations are extremely exuberant, decompensation may not occur during the entire disease. On the other hand, physical signs of a valvular lesion are usually frank and unmistakable. This is because of a pre-existing valvular lesion, and because the streptococcus viridans usually produces abundant valvular vegetations. Occasionally, when the mural instead of the valvular endocardium is chiefly involved, murmurs may be faint and not characteristic. Arrhythmias, especially auricular fibrillation and extrasystoles, are not uncommon. When found during the active febrile periods, they are apparently caused by endocardial, rather than by myocardial, involvement, for they usually cease with the disappearance of fever. When present during decompensation, cardiac irregularities are probably due to myocardial degeneration, or are indicative of myocardial insufficiency.

The study of blood cultures is naturally of extreme diagnostic importance. When properly made and a sufficient length of time (one week) is allowed for the appearance of the growth, the organism has been recovered in a great majority of cases. Sometimes numerous blood cultures are necessary before the organism can be isolated.

The clinical course of some cases in which the coccus has not been recovered during life, and the presence at necropsy of glomerular lesions characteristic of viridans infections, make it probable that some cases heal, or at least remain quiescent over a long period of time. The quiescent interval may last several years. These patients have been grouped as being in the "bacteria free stage" (Libman). The following cases are illustrative of this condition:

Male, aged 21, had scarlet fever, followed by severe endocarditis when seven years old. A double aortic and a mitral regurgitant lesion resulted. For many years there was no decompensation or other symptom except occasional attacks of hematuria. A few months before death the patient developed chills and fever; the streptococcus viridans was then found in the blood. In the light of our present knowledge this case seems to belong to the category just described. The intervals between the attacks of hematuria probably represent long remissions or "bacteria free stages."

Male, aged 40, entered the hospital with fever. He had a mitral and an aortic lesion. Blood cultures on two occasions showed the streptococcus viridans. The fever remained high for three weeks, during which time the patient was delirious. The temperature then gradually became normal.

The patient was not seen again for one year. In this interval he said that he had been quite well. He was readmitted to the hospital with pneumonia of embolic origin. The physical signs of valvular disease were the same as upon first admission. Blood cultures were negative. The patient made a good recovery from the pneumonia.

The extent and frequency of bacterial reinvasions (*i.e.*, the duration of the bacteria-free interval) are at least partly governed by certain physical characteristics of the vegetations. If the latter be covered by a firm, partially organized fibrin layer, bacteria are not as apt to invade the blood stream; while if the fibrin layer be thin or be so situated that the blood stream impinges with full force against it, the bacteria are much more likely to be washed into the blood current. The amount of fibrin covering the vegetations probably depends upon the virulence and the proteolytic digestive power of the invading organism.

Besides symptomatic treatment, therapy consists in the use of vaccines, preferably autogenous, and of horse serum sensitized to various strains of the streptococcus viridans. An occasional cure by these methods has been reported. In view of the exceptionally long remissions, sometimes for years, without the use of vaccines, final judgement regarding "cures" by serum or vaccine treatment should for the present be withheld. In my own cases I have observed no effect on the course of the disease from serum or vaccine therapy; nor was there any evident arrest of the pathological process. This was shown by the examination of such hearts at necropsy. For example, in one case in which the diagnosis of streptococcus viridans infection was established by blood culture four days after the initial chill, not only did autogenous vaccines and sensitized horse serum administered for months have no effect on the symptoms or course of the disease, but the postmortem examination showed exuberant and fresh masses of vegetation literally plastering the mitral valves, the left ventricle and left auricle. Unfortunately, therapy of all kinds, including intravenous injections of silver salts (Chapter XX), must at this time be regarded either as futile or as purely in the experimental stage. The wisdom of prophylactic vaccination against viridans infection in those cases in whom its later occurrence is feared deserves careful consideration. Blood transfusions are only of value in combating existent anemia. I have seen no good effects from transfusions in other stages of the disease, even when the blood is transfused from an immunized donor.

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## CHAPTER XVI

## CARDIAC SYPHILIS—AORTITIS—ANEURISMAL DILATATION OF THE AORTA—SACCULATED AORTIC ANEURISMS—NON-SYPHILITIC AORTIC ANEURISMS

Syphilis rarely attacks the heart during its secondary stage. A few instances however have been reported in which the myocardium alone was involved during this stage. These involvements may be sufficient to produce symptoms of myocardial insufficiency such as dyspnœa on exertion, indefinite precordial distress and pain, precordial sensitiveness to pressure, slight pretibial edema, sometimes also a faint systolic murmur at the apex.

The vast majority of cardiac syphilis occurs during the tertiary stage. The symptoms and pathological changes depend upon the degree, extent and admixture of muscular, valvular and endocardial involvement.

In occasional cases, isolated luetic valvulitis of the mitral or tricuspid has been found. In most instances, however, the aortic valves bear the brunt of the endocardial infection. In addition to the cusps, there is often widespread destruction of the aortic walls, finally resulting in aneurismal dilatation and in true aneurisms. Accompanying the aortitis are changes affecting the myocardium, the remainder of the endocardium, and the coronaries. Together they comprise a composite picture often similar to that found in non-luetic cardiosclerosis (Chapter XVII).

In the absence of a history of veneral infection or of other signs of syphilis, the presence of a strongly positive Wassermann blood reaction is of paramount importance in the diagnosis of cardiovascular syphilis. More than one blood examination may be necessary, or provocative injections of mercury or of small doses of salvarsan (arsphenamin) may be required to obtain a positive Wassermann reaction. If the blood Wassermann remains negative, the chemical and cytological examination of the spinal fluid may indicate the presence of syphilis, for it has been found that the spinal fluid sometimes shows definite evidence of syphilis when the blood does not. Thus tested by routine and other methods, the diagnosis of cardiovascular syphilis can be substantiated in the great majority of suspected cases. It must be remembered that cardiac syphilis is often a latent process and that cardiac manifestations usually occur only ten to twenty years after the initial lesion. Most cases therefore, are first seen by the physician in early middle life.

From the clinical standpoint, the characteristic symptomatology and signs of tertiary cardiac lues depend mainly upon the presence of aortitis alone, or upon aortitis as part of the picture of cardiosclerosis. It should be here remarked that syphilitic aortitis is not always accompanied by enlarge-

ment of the aorta. In the exceptional instances in which the myocardium alone is affected, the diagnosis usually rests on the ordinary manifestations of severe myocarditis. The presence of gummata in the myocardium must also be considered. They add to the possibility of the occurrence of a myomalaceous focus, or, when involving the conduction system, to the occurrence of heart block.

**Aneurismal Dilatations of the First Part and Arch of the Aorta.**—A century ago Hodgson made the fundamental distinction between sacculated and "dilatation aneurisms." He defined the latter as a "preternatural permanent enlargement of the cavity of an artery." The former is definite protrusion of only one side of its wall and may include a small or large part of the arterial caliber. This distinction is of clinical importance in aortic disease. The French still apply the term "Maladie de Hodgson" to aneurismal dilatations of the aorta associated with valvular insufficiency. Aortitis of the ascending and transverse aorta is now known to be of luetic origin in the great majority of instances. The pathology and mechanism of aneurismal dilatations of these parts of the aorta have been described in detail by Thoma, who had studied ninety-two cases. He ascribes their fundamental cause to lessened resistance to the arterial walls, and categorically states that "such weakened condition is not found in the descending thoracic aorta," which showed no change macroscopically.

The orthodiascopic picture of dilatation of the first portion and arch of the aorta has already been described (Chapter XII). On auscultation the normal first sound at the right base is accompanied or replaced by a soft blowing murmur, usually not transmitted beyond the first and second right interspaces. Occasionally the first sound is faint or absent. The second sound is accentuated, of varying quality, and is best described as bell-like, metallic or tinny. When typical it has a peculiar twang-like character, almost pathognomonic of dilatation of the first portion of the aorta. The first sound is often followed by a soft diastolic murmur, indicative of aortic insufficiency. The accentuated and metallic quality of the second sound is not always due to extreme hypertension, for it is frequently present with normal or very slightly raised systolic blood pressure. The factor most responsible for the abnormal second sound is, I believe, aortic dilatation which acts acoustically as a sounding box in reflecting and thus changing the character of the sound.

In marked aneurismal dilatation, especially of the arch of the aorta, there is often visible aortic pulsation in the jugulum. When not visible, it may be felt by insinuating the finger behind the manubrim sterni. In cases of aneurismal dilatation of the first part of the aorta, there may be more marked right than left carotid pulsation, a fact determined by placing the finger deeply behind the respective clavicles. At the apex, there is frequently a soft systolic murmur, probably due to relative mitral insufficiency; or there may be a loud murmur due to thickening of the mitral cusps and mural endocardium, with consequent regurgitation. If the aneurismal dilata-

tion is accompanied by left ventricular hypertrophy and by hypertension, there may also be a reduplicated apical impulse or a somewhat accentuated first, as well as a slightly metallic second sound at the apex.

**Aneurismal Dilatation of the Descending Thoracic Aorta.**—Reports of aortitis are almost entirely confined to disease of the ascending or transverse aorta, with fragmentary or no reference to the descending thoracic. McCrae, Allbutt, Osler and others consider aneurismal dilatation of the descending thoracic aorta extremely infrequent, a statement apparently based on postmortem findings, for no symptomatology was mentioned nor had a clinical diagnosis been made. I shall, however, indicate, through illustrative cases the comparative frequency, diagnosis and symptomatology of aneurismal dilatations of the descending aorta, and present its claim to a clinical entity.

W. W., male, aged 69, has never been seriously ill previous to his present complaint. He had been a heavy smoker and had suffered from a venereal infection forty years ago, for which he had received many subcutaneous injections (presumably of mercury). His present illness began two years ago with exceedingly mild symptoms: Very slight precordial pains when lying on the left side, and slight dyspnœa when climbing stairs. At examination, the patient looked well preserved, the carotid pulsation on both sides was somewhat exaggerated, there was a belt of small dilated capillaries over the lower part of the chest. The cardiac thrust at the apex and the systolic impact at the right base were somewhat exaggerated. The cardiac area seemed normal to percussion. At the base there was a double murmur—a rough systolic and a softer diastolic—transmitted and best heard to the left of the middle third of the sternum; a definite sensation of heaving impulse was imparted to the examining hand placed over the same area. Similar but fainter murmurs were heard at the apex. Both radial pulses were alike; the average systolic blood pressure was 170 m.m., the diastolic 70. Neurological and other examinations revealed no abnormalities. There was slight pretibial edema. The Wassermann reaction was negative. The roentgenogram (Fig. 264) showed a long fusiform dilatation of the entire descending thoracic aorta. The electrocardiogram presented evidence of left ventricular preponderance, *i.e.*, negative S III. Treatment consisted of one salvarsan and many mercurial injections; iodide of potash in moderate doses was administered in alternate periods of two weeks. Tincture of digitalis was given for one week until edema of the legs disappeared. The patient has been under my observation for many years and is quite comfortable, with no pains for the last three years; the physical signs along the middle third of the sternum have markedly receded.

Female, aged 47 years, married sixteen years, had never been pregnant. Prior to the present illness she complained of dyspnœa on exertion, and of nocturnal palpitation accompanied by pain in the lower precordium. She had lost about forty pounds since her illness began. One week before admis-

sion to the hospital her legs became edematous. The patient was emaciated, the right pupil was somewhat larger than the left, both reacted sluggishly to light but normally to accommodation; the knee reflexes were diminished; Romberg's sign was absent. The patient was dyspnœic even when at rest. There was vigorous visible carotid and jugular pulsation; the aortic thrust could be plainly felt by pressing the finger tip behind the manubrium. By placing the eye on a level with the patient's chest, two distinct areas of impact were discernible; one corresponding to the apical region, the other to an area slightly to the left of the lower half of the sternum. The latter impact was also palpable by sinking the fingers in the lower left intercostal spaces, and also by auscultating with the stethoscope over this area in the routine manner. There was no precordial tenderness. The apex was felt most distinctly in the sixth interspace, a considerable distance (14 c.m.) from the midsternal line. Over the base, and particularly to the left of the midsternum, there was a rough systolic murmur and a somewhat accentuated second sound merging into a soft murmur occupying the entire diastole. Friction sounds indicative of dry pericarditis were present at the base and apex. There was slight edema of the legs. The Wassermann reaction was negative upon first examination; some months later it became positive. The systolic blood pressure ranged between 200 and 180 m.m., the diastolic between 100 and 40 m.m., the pressure in both arteries was equal. The urine was normal. The roentgenogram (Fig. 265) showed fusiform aneurismal dilatation of the entire descending thoracic aorta; orthodiascopic examination corroborated this finding. The electrocardiogram presented evidence of left ventricular preponderance. The patient refused treatment. She has since reentered the hospital, with signs of severe cardiac failure. She received iodide of potash and mercurial injections, with marked improvement.

Male, aged 38, a vigorous and healthy looking man, has complained during the last six months of slight dyspnœa upon climbing stairs, but none when at rest. He was a heavy cigar smoker. He had gonorrhea twenty years ago and denied any other illness. The blood pressure was normal. There was vigorous carotid pulsation at the root of the neck. There was no pain on precordial pressure. The apex beat was strong and was felt best in the fourth interspace, 11 c.m. from the midsternal line. A soft systolic murmur was heard at the apex. At the base the first sound was impure; the second sound was not accentuated but was prolonged and somewhat liquid in character, and occupied the entire diastole. These abnormal sounds were heard loudest and most distinctly along the middle left sternal border. The eye placed on a level with the chest could discern a slight systolic heave over the latter area, apparently distinct from that at the apex. The other organs were normal. The Wassermann reaction was negative. The roentgenogram showed dilatation of the upper part of the thoracic aorta, the latter being visible as a somewhat denser shadow behind the ventricles. The orthodiascope confirmed this. The electrocardiogram was normal. The

patient was given salvarsan, 0.3 gms. intravenously, and many bichloride injections combined with iodide of potash. The dyspnœa disappeared entirely; the abnormal sounds at the base and left sternum were much less pronounced than at the first examination.

**Differential Criteria of Aneurismal Dilatation of the Descending Thoracic Aorta.**—It is important to distinguish the aortic impacts to the left of the sternum from those found in patients with marked left ventricular hypertrophy or in healthy individuals with over-acting hearts and thin chest walls. In these individuals, the auscultatory signs above described are then absent. Although there are many refinements of percussion methods used in the attempt to delicately outline the dilated arch and the ascending aorta (for example, threshold and auscultatory percussion, Chapter XIII), their value in dilatation of the descending aorta seems nil because the aorta is deep seated and most of the enlarged area is situated behind the ventricles. Examination of the posterior chest wall also fails to reveal any difference from the normal physical signs. In three of my cases in whom a tentative diagnosis was made before fluoroscopy, all methods of percussion failed to reveal any enlargement of the descending aorta. To clinch the diagnosis, examinations by means of the fluoroscope, or roentgenograms are essential. As in examination for suspected disease of other portions of the aorta, the patient should be fluoroscoped in several lateral positions. Fluoroscopy must be practiced carefully in order to reveal and distinguish the darker silhouette of the dilated descending aorta behind the left ventricle. Roentgen-ray plates must also be carefully scrutinized for the same reason. It is important in this connection to again indicate the difference in shadow areas between orthodiascopic fluoroscopy and roentgenograms of the heart and aorta. It has been my experience that the former produces very little distortion in the size of the cardiac and aortic areas, because the X-ray tube and screen move together (Chapter XII), thus approximately parallel rays reach the observer. In the roentgenograms the rays are always divergent and there is an increased cardiac shadow. Comparisons between the orthodiascopic tracings and roentgenograms in my series of cases occasionally showed marked discrepancies in the size of the dilated aorta and heart by these two methods.

Because of its importance I shall summarize the clinical complex of aneurismal dilatation of the descending aorta as I have observed it. The Wassermann test was done in four of the five cases. It was positive in two and negative in two. One of the latter gave a definite history of luetic infection. Three cases had slight symptoms when treatment was begun; these were clinically cured. One, with severe heart failure, was much improved. In three the correct diagnosis was made by the presence of an impact area to the left of the sternum at its middle third, and by prominent localization of the murmurs over this area. Electrocardiograms were taken in four cases; three showed complexes of left ventricular preponderance, the fourth was normal. The physical signs of thc cases were most marked when the symp-

toms—dyspnœa, pain, or cardiac failure—were present; the signs became markedly less with improvement.

**Symptomatology of Aortic Dilatations—Aortitis.**—There is no distinguishing characteristic in symptomatology between dilatation of the various aortic divisions: They are therefore grouped in this description. Sharp, continuous, gnawing pains such as those often associated with sacculated aneurisms are not prominent features of dilatation of the descending aorta. When present, substernal pains or those referred to different parts of the chest, neck or head are most apt to occur with exercise or following physchic and emotional disturbances. It is difficult to state the exact etiology of these pains. They are probably not due to pressure of the dilated aorta upon the surrounding structures (esophagus, ribs, dorsal vertebra, intercostal nerves, etc.). In one of my cases, pain may have been due to esophageal stricture rather than to aortic pressure. In general the contour of the aneurismal dilatation would in itself argue against pressure upon, or erosion of surrounding tissues. The rich nerve and ganglionic plexus surrounding the root of the aorta, and the nerve fibers and isolated nerve cells which have been found in the connective tissue of its middle coat, may explain how various grades of inflammation in the aorta and how differences of aortic pressure and dilatation can give rise to these referred pains. In addition, peri-aortitic inflammation with possible involvement of the neighboring nerve structures can also cause neuralgic symptoms. Similar nerve involvement has been found in dilatations of the ascending aorta and arch. The assumption of inflammatory exacerbations within or without the aorta is corroborated by the occasional rapid subsidence of the pains following salvarsan injections. This result is probably ascribable to control of these inflammatory recurrences, although Vaquez and Laubry, and Vaquez and Bordet claim that there is sometimes a reduction in size of sacculated aneurisms after several salvarsan injections. The writer has not been able to determine any difference in the size of the dilated aorta as the result of therapy. In addition to the aortic disease it must be remembered that concomitant coronary sclerosis and myocarditis can also produce cardiac pains. Another causal factor of pain is involvement of the coronaries in the luetic process. Precordial pain from this source is similar to that usually found in coronary disease (Chapter XXIII). Sudden overdistention of the ventricle from increase in intraventricular pressure by blood regurgitated from the aorta may be another cause of severe attacks of precordial distress. It is conceivable also that the coronary supply may be mechanically interfered with by tension of the diseased and dilated aorta, this again giving rise to precordial or substernal pain.

Dyspnœa is a common symptom, especially in advanced aortitis, even without extensive myocardial changes. It is often paroxysmal and nocturnal in character. Some of these attacks may be due to pulmonary congestion or edema. Others are undoubtedly of an entirely different nature; it is to these that I wish to call particular attention. They have already been emphasized

by others (Longcope, Lamb). These attacks are neither pulmonary nor cardiovascular but are in the nature of bronchial asthma. Indeed, the physical signs are exactly those of the latter condition. There is an emphysematous note on percussion; there is typical sibilant and sonorous breathing, there is wheezing inspiration and prolonged expiration. The two subjoined cases well illustrate the clinical picture.

M. C., aged 38, married and father of several healthy children, entered the hospital with what appeared to be a foudroyant attack of typical bronchial asthma: There was sibilant and sonorous breathing, with an emphysematous percussion note over the entire chest. Dyspnœa was extreme and alarmingly violent. The heart sounds could not be accurately made out because of the difficulty in breathing. Nonetheless definite systolic and diastolic murmurs could be heard over the right base. The next day there was skin crepitation over the entire chest from rupture of the pulmonary vesicles. The Wassermann blood reaction was 4 plus. The skin emphysema rapidly disappeared. The asthmatic condition only slowly receded during the patient's hospital stay of several months, despite vigorous antiluetic treatment, iodides, adrenalin and atropine. With partial recession of the asthma, there were typical signs of a luetic aortic lesion with aortic regurgitation. The X-ray showed but slight enlargement of the arch of the aorta. The patient has been under my observation for several months subsequent to his discharge from the hospital. At no time were *cardio-vascular* symptoms present. Therapy indeed was almost continuously directed to the asthma. The usual mixture given contained iodide of potash, tincture of belladona and chloral hydrate. When sibilant breathing and wheezing were not extensive or marked, the patient's condition was comfortable, there was correspondingly less dyspnœa and he was partially able to attend to his work as a piano maker. When asthma was more prominent, breathing was again difficult. Of late months, the asthma has gradually subsided, so that for the present at least the patient is practically symptom-free although the physical and X-ray signs of the aortitis are exactly as before.

A. F., male, aged 52, a flutist, was always very active physically, and prided himself upon his physical stamina. Eight months prior to my first examination, he was suddenly attacked with violent cough, oppression and pain in the chest. Since then the main complaints have been cough and some substernal discomfort especially when walking. Examination revealed the signs of aortic dilatation over the right base, a rough systolic and a diastolic murmur. This was confirmed by X-ray, which showed aneurismal dilatation of the first portion of the arch of the aorta. Further physical examination of the chest showed typical emphysematous and asthmatic breathing, there was no evidence of pulmonary congestion or edema. The patient was given an asthmatic mixture containing iodide of potash and tincture of belladonna. Intramuscular mercurial injections and intravenous neosalvarsan injections were also begun. Within two weeks there was marked improve-

ment in his condition. The sibilant breathing had almost entirely disappeared, he could walk two miles fairly comfortably in windy weather along a country road. At the end of two months, the sibilant and asthmatic condition had disappeared, the patient considered himself well. X-ray and physical examination of the chest however revealed the same condition of the heart and aneurism as before.

These two cases give undubitable proof, I believe, of a distinctly bronchial asthmatic type, but have no reference to the size of the aortic dilatation; for in one case, the aorta was practically normal in size (and this was the severe case) and in the other there was the usual marked aneurismal dilatation. Nor have these cases any direct relation with assumed pulmonary stasis or with a tendency to heart failure. Both of the patients, for example, considered themselves well when the asthmatic condition came under control, although the cardiac condition remained the same. It seems that instances of bronchial asthma with aortitis are explicable only upon the supposition of a bronchial spasm resulting from reflex disturbances initiated in a diseased aorta. It has already been pointed out that the aorta is surrounded by many nerve plexuses and ganglia (Chapter I). There exists experimental evidence that certain manipulation of the inner surface of the aorta may also evoke this reflex, with resultant bronchial spasm. These facts and the clinical evidence make it highly probable that disturbance of innervation by aortic disease can likewise produce a spasmodic asthmatic condition in the human being. In luetic aortitis, the primary instigating factor seems to be a periaortitis, which when controlled or controllable by proper remedies, causes recession of the bronchial spasm and relief of the asthma.

Another group of symptoms of aneurismal dilatations is that due to cardiac decompensation. This is not necessarily a marked clinical feature; in fact, it may only consist of slight dyspnœa upon exertion. Its presence seems due to accompanying cardio-vascular disease rather than to the aortic dilatation itself. Edema is usually slight and confined to the legs; it is extreme only late in the disease or in neglected cases. Dyspnœa is of the usual cardiac type; it is generally most marked on exertion; it is continuous in the severe cases with cardiac failure. Left ventricular hypertrophy of varying degrees is usually present. The heart is occasionally hypertrophied before cardiac failure sets in. It is a mistake, however, to assume that ventricular hypertrophy is present in every case of syphilitic aortitis, for occasionally the cardiac chambers are not appreciably enlarged.

**Sacculated Aneurisms.**—As already mentioned, the physical characteristic of sacculated as distinguished from dilatation aneurisms is that the former is a protrusion of "only one side of the caliber of an artery and may include a small or large part of the arterial caliber." The pathological process is similar to that found in dilatation aneurisms except that the damage is not general but is localized in portions of the artery, thus causing localized weakness of the arterial coats, with gradual bulging and the final formation

of a sac. The site of the aorta most frequently affected is its first or ascending portion, but any part including the abdominal aorta may become the site of a sacculated aneurism. Although one sac is the rule, as many as nine different sacs have been described. Depending chiefly upon the area and degrees of weakness of the arterial wall, the aneurismal sac may have the most diversified shape and size. It may be saucershaped, pearshaped, oval, pedunculated, circular, etc. It may be so large as to occupy an entire half of the chest.

The physical signs to some extent resemble those of dilatation aneurisms and are indeed sometimes identical with them. Depending chiefly upon such gross characteristics as the size of the sac and its nearness or adherence to the chest wall, thrills, murmurs and visible pulsations are apt to be more prominent than in dilatation aneurisms. Percussion may be of value if the sac is near to the chest wall. It is important to emphasize, however, that sacculated aneurisms, even fairly large ones, may give rise to no physical signs at all, and unless the patient is fluoroscoped, the diagnosis may be missed.

It is clear that an aneurism which has eroded bony and soft structures, or has pushed the latter aside so that the sac forms an easily visible pulsating tumor, can be readily diagnosed. Our diagnostic difficulties occur with deeper sacs with unclear, masked, and equivocal physical signs, so that either the aneurism is not suspected, or if suspected, it may be difficult to distinguish it from a mediastinal or pulmonary tumor. In all instances when examining for aneurisms, inspection of the bared chest is of special importance. This is usually best practiced with the patient in a recumbent position, in a good light, and the observer's eye on a level with the chest wall. In this manner pathological pulsatile heaves of various portions of the aorta in its relation to the chest wall may be distinguished. It is of course necessary to be familiar with the normal impacts and pulsations, especially those due to the aorta and left ventricle (Chapter XIII) in order to diagnose those abnormal ones caused by a sacculated aneurism. It is also necessary to scan the patient's back carefully, for occasionally the aneurism bulges posteriorly. Palpation should be as carefully practiced as inspection, especially over the assumed site of the tumor. This is best carried out by pressing the flattened fingers well into the intercostal spaces. Auscultation usually reveals a rough systolic murmur over the pulsatile area, especially if the tumor be large and near the chest wall. Under such circumstances, the murmur is also plainly audible in the carotids. Depending upon the size of the sac and the degree of dilatation of the aortic ring, there may be an accentuated or twang-like second sound, or a variably distinct diastolic murmur. In one instance that I observed—an aneurism of the transverse arch protruding into the anterior chest wall—there was a reduplicated second sound to be heard and felt, apparently due to asynchronous closure of the aortic cusps.

The heart may be pushed laterally or downward, depending upon the size and direction of growth of the aneurismal sac. Such misplacement is

often mistaken for cardiac hypertrophy. Hypertrophy however, is by no means invariable with aneurism; the sac indeed may be very large, and the heart itself be normal in size or only slightly hypertrophied.

In addition to the physical evidence of sacculated aneurism derived as above outlined, there are other concomitant signs which may help to clinch the diagnosis, or make us suspect aneurism when more direct signs are lacking. Tracheal tug is frequently mentioned as one of these. It is by no means invariably present and is found only when the sac is adherent to the trachea. Over-pulsatile carotids are frequently associated with sacculated aneurisms: They are of course present in many other abnormal cardiac conditions (Chapter XIII). If by palpation and inspection it is determined that there is a distinct difference in the carotid throb on both sides of the neck, importance is lent to the probability of aneurism. Inequality in the radial pulses and in brachial blood pressure is common in arotic aneurisms. Usually the right radial is smaller than the left because of involvement and narrowing of the innominate artery. Inequality of the pupils is fairly constant. Suffusion of the facies and swelling of one or both upper extremities may occur as the result of pressure upon the veins at the root of the neck: This is usually a late sign. The superficial venules and capillaries of the skin of the chest may be dilated for a similar pressure reason.

In modern practice, no examination for suspected aneurism is complete without an X-ray examination. An X-ray plate should always be supplemented where possible by a careful fluoroscopic examination. For instance, a photograph or plate may show only a questionable enlargement of some portion of the aorta, while fluoroscopy may show overpulsatile expansion and excursion of the suspected area, thus aiding in clinching the diagnosis. The patient should be fluoroscoped in several lateral positions in order to render all parts of the aorta visible.

**Symptomatology of Sacculated Aortic Aneurisms.**—Small aneurisms that do not cause pressure are not apt to give rise to symptoms. Inflammatory exacerbations (peri-aortitis) may give rise to pain, especially sub-sternal in distribution. Concomitant coronary disease is another frequent cause of pain. Pressure against various nerve and bone structures, especially in aneurisms with erosive tendencies, naturally cause pains of various distribution. Finally, continuous and excruciating pains may be present. Pressure upon the phrenic nerve may produce hiccough; upon the sympathetic, unilateral dilatation of the pupil, and sweating and flushing of the face. Pressure upon the recurrent laryngeal nerve as it curves around the aortic arch usually produces a brassy or croupy cough; in other cases the voice is altered so that it sounds cracked. Pressure against the esophagus produces dysphagia; ultimately ulceration of the esophagus may result. Pressure against the trachea and bronchi, when mild, gives rise to an irritating cough; with severe pressure, there may be actual bronchial stenosis, a condition which may mask the actual disease.

**Therapy of Luetic Aortitis.**—What follows under this heading applies to all types of aortic syphilis, aortitis, and aortic dilatations with and without sacs. Treatment may be conveniently divided into three parts: That of the underlying disease, of the decompensation, and of the pains. Salvarsan, (arsphenamin) was originally considered contraindicated by Ehrlich in cardiac lues because of the fear of overwhelming the system with spirochetes (Herxheimer reaction); but experience has shown that the drug judiciously administered is definitely indicated in cardiac syphilis. The safest method is to begin with several intramuscular injections of mercury and the administration of iodide of potash. This is followed by intravenous injections of arsenic compounds. In the majority of cases, the best routine procedure is the intravenous injection of 0.2 gms. of salvarsan or arsenphenamin (or 0.45 of neosalvarsan) every ten days until three doses have been given; then if indicated, it may be repeated in 0.6 gms. doses a month or two apart. A course of several intravenous injections is again followed by mercurial injections and the iodides. Usually these procedures render a previous plus Wassermann negative. If the luetic changes in the aorta are such that calcification and scar-tissue formation are extensive, and the myocardium is the seat of advanced disease, the treatment can naturally be of little or no avail. The degree and extent of such pathological changes cannot be diagnosed accurately enough by our present methods, although Vaquez and Bordet claim to have observed with the fluoroscope calcified areas in the ascending and transverse aorta, and the diminution of these areas after salvarsan injections. Since the treatment outlined is often efficacious and is followed by marked improvement, I believe it should be carried out in all cases unless there be marked heart failure. When luetic aortitis is strongly suspected, even if the Wassermann reaction is negative, the same therapy should be instituted, because in any case salvarsan is only rarely followed by serious results. An occasional case, however, may be encountered in which even small doses of arsenphenamin, carefully given, do damage, apparently by lighting up dormant pathological processes.

Cardiac failure requires the same treatment as that from any other cause. A reliable preparation of digitalis should be given (Chapter XX). I prefer digitan tablets or the tincture administered undiluted in 15-drop doses three times daily. If the case be urgent, and very little or no digitalis had previously been given, 1 c.c. of a 1 per cent. solution of strophanthin can be slowly injected intravenously. The objection that digitalis in therapeutic doses raises blood pressure has been sufficiently disproved by many careful investigations (Chapter XX). Digitalis had no effect upon the blood pressure in any of my cases; the pressure was as often lowered as raised during its administration. Symptomatic treatment of the pains occasionally requires codein or morphine, but pains are frequently relieved by the antiluetic treatment which acts by controlling the inflammatory exacerbations of aortitis and periaortitis.

**Prognosis.**—Patients with aneurismal dilatation who seek advice before severe decompensation sets in, and in whom vigorous anti-luetic treatment can be instituted, may live in comparative comfort, for the aortic disease is an index of the general cardiovascular mischief rather than in itself the cause of cardiac failure. In those with sacculated aneurisms, the ultimate prognosis depends largely upon the size of the sac, its adherent and erosive tendencies, and the pressure it produces. When the sacs are large, protuberant and adherent to the chest wall, the patients usually live, at the utmost, but a few years; in exceptional instances, however, many years may elapse before death ensues.

Cure, in the sense of a return of the aorta to its normal state, is impossible; but, as in other organs, the luetic disease can be arrested and controlled, so the heart and aorta, although still crippled, may be sufficiently restored to make the patient comfortable. Rupture of a dilatation aneurism is extremely infrequent. Unless the luetic disease is confined to a small area, this accident seems less likely to occur in the descending aorta because of its greater length and because the dilatation occupies a larger area. In the sacculated varieties, one massive bleeding, or smaller hemorrhages through the skin or into the bronchus are by no means rare. Hemothorax, caused by aneurismal rupture into the pleura is another occasional late accident.

Mention should be made of various operative procedures intended to obliterate the sac, especially the introduction of wire. The object is to have layers of fibrin deposits from the blood clot separate out, thus diminishing the calibre of the artery. A recent method has been to introduce very fine wire into the sac, and then to send an electric current through the wire in order to induce electrolysis. I have observed two cases treated by this method. In one, the end of the wire perforated the chest wall and gave rise to hemorrhages from which the patient finally succumbed. The other lived several months after the operation.

**Aortic Aneurism and Aneurismal Dilatation Due to Rheumatism.**—Klotz in particular has called attention to the possibility of rheumatism as an etiological factor of aortic aneurism. When aortic aneurism is present in children, an occasional occurrence, congenital syphilis must be definitely excluded before rheumatism is considered in a causal relation. However, the role of articular rheumatism as the causative factor of aortic aneurisms in the middle-aged and old has perhaps received insufficient consideration, because attention is usually focussed upon frank valvular defects in those with a rheumatic history and because in the arterio-sclerotic aneurisms of the old (q. v.), a definite and unmistakable rheumatic history is long since forgotten or overlooked. In occasional instances, however, the clinical correlation between articular rheumatism and aortic aneurism seems unmistakable. The following is an example.

Mrs. R. D., aged 65, no history of any serious acute illness until thirteen years ago. She then had a typical attack of inflammatory rheumatism last-

ing several weeks. There were no cardiac symptoms until one year ago, when her present complaints began. These are chiefly dyspnœa, tachycardia and occasional pains in the left arm. Examination revealed a systolic blood pressure of 160, diastolic, 100. There was a palpable thrill over the right base. There was hyperactivity of the carotids and of the aorta in the jugulm. There was a rough systolic murmur and a slightly accentuated second sound over the right base, and a rough systolic murmur over the mitral area. The X-ray showed marked aneurismal dilatation of the first portion and arch of the aorta. The Wassermann reaction was negative.

This case emphasizes the fact that rheumatism can occasionally act as a vascular and not only as a valvular poison.

The physical signs, pathological picture and symptoms of rheumatic aortic aneurisms and aneurismal dilatation are no different from those of arterio-sclerosis aneurisms. (See below.)

**Atheromatous and Arterio-sclerotic Aneurisms and Aneurismal Dilatations.**—These conditions are by no means unusual in the old and senile. They rarely occur without associated cardio-sclerosis (Chapter XVII). The murmurs they cause are sometimes quite loud and are heard over large areas. The diseased process first attacks the intimal coat and produces various degrees of arterial thickening, and in advanced cases, calcified placques. At this stage the aorta is not necessarily enlarged. It is only when the media becomes affected that, similar to aortic syphilis, the aortic walls weaken and give rise to aneurisms and aneurismal dilatations. These do not as a rule reach the enormous size of luetic aneurisms, although I have seen some in the X-ray which involved the first portion and arch of the aorta and reached considerable proportions. The physical signs are those of syphilitic aortitis (q. v.) except that the rather distinctive twang-like second sound at the right base is usually absent. The condition is commonly only part and parcel of cardio-sclerosis (Chapter XVII), hence generalized arterio-sclerosis is a frequently concomitant.

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## CHAPTER XVII

## ACUTE MYOCARDITIS—SYMPTOMATOLOGY, PHYSICAL SIGNS, DIAGNOSIS AND PROGNOSIS OF CHRONIC MYOCARDITIS AND CARDIO-SCLEROSIS—THE HEART IN CHRONIC EMPHYSEMA—FATTY HEART

Because the pathological changes found in myocarditis vary considerably in extent and degree (Chapter IV), it becomes apparent that its symptomatology must be a varied one, and to a great extent, must depend upon the type and extent of the pathological process.

**Acute Myocarditis.**—It should be emphasized that while many infectious diseases are undoubtedly accompanied by some inflammatory change in the myocardium, chiefly cloudy swelling (Chapter IV), such changes are so fleeting in character or are so slight, that they give no clinical evidence of their existence. Hence myocardial damage found at postmortem, unless widespread and severe, does not necessarily imply that the patient suffered from the clinical evidences of myocarditis during life. On the other hand, cardiac death may be the result of a severe toxemia as in pneumonia, yet the heart may show comparatively little damage at autopsy, either macroscopically or microscopically. Such and similar instances no doubt rest upon the fact that important cardiac *functions* (especially contractility and conductivity) may be interfered with by poisons elaborated by various diseases during life and yet comparable actual visible pathological changes may not be found postmortem. It has already been indicated in various connections (Chapter V) that some diseases (especially rheumatism and diphtheria) are particularly apt to be accompanied by clinical evidence of acute myocarditis. The clinical symptomatology and signs of these conditions have been elsewhere described (Chapters XI, XXVI).

Acute myocarditis is probably extremely rare as a primary affection. It is usually secondary to endo- and peri-carditis and to invasion of the blood stream by bacteria and toxins.

**Chronic Myocarditis.**—The pathological features of chronic myocarditis and cardio-sclerosis have already been detailed (Chapter IV). Chronic myocarditis as an *isolated* lesion is very rare; and its diagnosis as an isolated lesion may be extremely difficult or perhaps impossible. The presence of definitely correlated infectious factors in the patient's history, especially acute rheumatism and diphtheria, aid considerably in the diagnosis.

The physical signs of chronic myocarditis as an isolated entity may be limited to some definite evidence of enlargement of the heart, shown prefer-

ably by X-ray rather than by percussion. In advanced cases with severe degeneration, there may be a weakened and diffuse apical impulse, as well as weakness of the first sound at the base and apex, and weakness or entire absence of the second sound, especially at the base. However, it may not be possible to venture more than a guess as to the extent, or even the presence, of any myocardial damage from the physical examination alone. But when the *entire* clinical picture is correlated and considered, with its positive and negative data:—a definite etiological factor; an increase in the size of the heart; normal palpable arteries; absence of valvular disease; the presence of muffled and indistinct cardiac sounds; moderate tachycardia and dyspnœa; electrocardiograms indicative of myocarditis (Chapter IX); the subjective and objective phenomena indicative of myocardial insufficiency and heart failure (Chapter XIV); then, it may be possible to diagnose with a fair degree of accuracy not only the presence of chronic myocarditis as an isolated lesion, but even the extent of myocardial mischief. On the whole, it is clear that the diagnosis must often be established inferentially and by exclusion.

**Cardio-sclerosis,** also called the arterio-sclerotic and senile heart, presents a composite pathological picture (Chapter V) in which chronic myocarditis of varying degrees is almost always present.

Although the general subject of arterio-sclerosis is beyond our scope, it is necessary to indicate its importance in cardio-sclerosis. While arteriosclerosis is usually general, insufficient emphasis is laid upon the fact that it is often an arteriolar rather than an arterial disease, and that the arterioles are by no means equally diseased throughout the cardio-vascular-renal system. We do not know the cause of this uneven distribution of the diseased process, but it is upon this basis of selection that one organ rather than another, presents the predominantly clinical syndrome. Indeed, arteriolar disease may be so selective that the coronaries alone may bear the chief clinical brunt of the disease. If the process be confined mainly to the smaller arteries of the brain, symptoms and dangers become chiefly cerebral: if in the coronaries, the pathology and symptomatology is that of cardio-sclerosis; if in the coronaries and aorta, the pathology and symptomatology is that of aortitis and cardio-sclerosis; if in the kidneys, especially in the glomeruli, varying types of renal sclerosis with their symptomatology come prominently in the foreground; if in the retinal vessels, there are corresponding ophthalmological changes. It may be mentioned parenthetically that careful examination of the eye backgrounds often offers corroborative, indeed sometimes the only *objective* diagnostic proof of the probable existence of more or less generalized arterial or arteriolar disease. This fact does not usually receive the clinical importance it deserves. This brief summary of selective vascular disease also indicates the frequent clinical overlapping of the diseased process in the various vital parts of the cardio-vascular renal system. Often it tries the skill of even a broadly trained clinician to decipher and properly group the complex clinical picture and to apportion the pathological damage.

**Physical Signs.**—If cardio-sclerosis is at all advanced, it is readily diagnosed from physical signs. Prominent among these are the signs of aortitis. If the latter be accompanied by simple or aneurismal dilatation of the arch of the aorta (Chapter XVI), there may be on inspection, a broad expansile rise of the tissues in the suprasternal notch, due to the vigorous overaction and enlargement of the arch. In advanced cases of arteriosclerosis, not only the radial and temporal arteries but also the carotid vessels may be tortuous and thickened, and may pulsate very vigorously with an accentuated thrust. If the first part of the aorta be especially involved in the atheromatous process, this carotid phenomenon may be more marked on the right than on the left side. In a thin chested individual, with the patient lying prone, and with the eye of the observer on the level with the patient's chest, it is sometimes possible to see pulsatile rises of the tissues over the right second interspace in simple or aneurismal dilatation of the aorta.

Corresponding to the physical signs discovered by inspection, on palpation, one can feel a marked thrust by lightly insinuating the finger behind the sternum; a similar thrust may be found over the carotids. Systolic expansion over the right base may likewise be felt by snugly applying the fingers laid flatly in the second and third right interspaces near the sternum.

The auscultatory signs of typical aortitis are characteristic and readily recognized. The first sound is rough and harsh; the second, accentuated and in some instances, metallic and tinny in character. The latter has usually been interpreted as due to hypertension, a frequent accompaniment of aortitis. It is fairly frequent, however, when hypertension is not present. The roughened and harsh murmur over the right base is sometimes transmitted to the aortic arch and may be plainly heard by placing the bell of the stethoscope in the jugulum over the pulsating arch; transmission of the murmur depends partly on the extent of the aortic enlargement and partly on the height of the arch. The murmur is likewise occasionally propagated along the carotids. Following the second sound, a diastolic murmur of varying duration and intensity may be heard over the right base. It is usually transmitted downwards along the right sternal border; on rare occasions it is heard best over the third interspace and along the left sternal border.

Although the physical signs above described are characteristic and typical of aortitis, there is scarcely a cardiac lesion which produces more varied or atypical auscultatory phenomena. For example, the sounds may be perfectly normal at the right base, the first or second sound, or both may be extremely faint or entirely absent. Upon what changes in the aorta or in the heart itself such differences depend it is at present impossible to state.

The sounds that are heard at the apex are rarely significant in the diagnosis of aortitis. They apparently vary with the state of efficiency of the circulation, and, to a lesser extent, with the state of hypertension. For example, the first sound may be muffled or scarcely audible. Occasionally, in hyperten-

sion with or without left ventricular hypertrophy, the second sound at the apex takes on characteristics similar to that at the right base, that is, it becomes metallic in tone. In conjunction with a weak or absent first sound, the cardiac impact also may be weak or scarcely palpable. I was able to prove at necropsy one instance characteristic of these findings. The patient complained of severe epigastric pains, especially on walking. The cardiac examination showed a muffled first sound over the right base and a scarcely perceptible first sound at the apex. Polygraphic tracings showed heart block. The orthodiascopic tracing showed enlargement of the first part and arch of the aorta, and an enlarged left ventricle. The patient died suddenly. At necropsy, there was diffuse dilatation and thickening of the aorta up to, and including the upper abdominal aorta; spirochetes were isolated from the aortic wall. There was moderate left ventricular hypertrophy; the myocardium was riddled with scar tissue.

While physical signs like those described are indicative of myocarditis and aortitis, these lesions can be present and yet sounds at the apex and base be perfectly normal. A faint systolic murmur may accompany the first sound; this, however, possesses no diagnostic value, for such impurities are by no means rare in normal hearts (Chapter XIII). Of much more significance is the presence of a loud, rough systolic murmur heard most prominently at the apex and sometimes transmitted to the left. These murmurs are probably caused by fibrous or calcareous valvular thickening with consequent mitral regurgitation. In old individuals with marked senile arteriosclerosis and widespread aortic and valvular changes—a cardiovascular condition also found occasionally in the middle-aged—a very loud, rough, systolic murmur is sometimes heard over the entire anterior part of the chest. This murmur is transmitted to the left, and may even be heard posteriorly between the upper part of the scapulæ. In its harshness, loudness and area of transmission, it is extremely suggestive of aneurism. However, a diastolic murmur is rarely encountered and the roentgenograms of the cases that I have studied have not regularly shown abnormal aortic enlargement. This loud systolic murmur is apparently the result of the coalescence of two components: An aortic systolic and a mitral regurgitant murmur. The aortic factor is due either to atheromatous change in the aortic valves producing stenosis, or to the impact of the impinging blood stream over the roughened sclerotic aortic wall; both causes sometimes operate together to produce this aortic component. The mitral component of the murmur is due to sclerotic changes in the mitral cusps. The intensity of the combined murmur seems to depend on the state of myocardial efficiency. I have found it exceptionally loud only in those who were compensated, or in whom there was but slight cardiac failure. The mechanism of the murmur with illustrative cases is described in detail in Cardio-Vascular Clinics. On the other hand, a murmur may be absent in those who clinically or at necropsy show advanced cardio-sclerotic changes. Such an instance is the following:

A robust man of 44 complained chiefly of substernal pains upon slight exertion. There were occasional attacks of nocturnal dyspnœa. The only abnormality upon auscultation was slight muffling of all the cardiac sounds. Several Wassermann blood tests were negative. The patient finally died of heart failure. At necropsy, encrusted calcareous deposits were found on all the valves. The coronaries were thickened and impermeable, the aortic arch was markedly atheromatous, the myocardium contained many fibrous patches. As already suggested, the absence of loud, harsh murmurs in this case was probably due to failing circulation.

An extremely valuable hint regarding the degree of cardio-sclerotic change is sometimes given by the presence of pericardial adhesions. These are found most frequently over the right base or in the apical region. Superficial creaking sounds or pericardial rubs are then heard at the end of long inspiration, or with cardiac systole. The importance of diagnosing these old localized adhesions lies not so much in the fact that they add embarrassment to the circulation, but because they are found only when the cardio-sclerotic changes are advanced. Some of these adhesions may be due to localized pericarditis covering myocardial infarcts from advanced coronary disease.

Thus far, most stress in the diagnosis of cardio-sclerosis has been laid upon the auscultatory signs. Other important diagnostic data may be gained by examination for ventricular hypertrophy, hypertension, and palpable and audible reduplicated first sounds at the apex. The significance and bearing of all these phenomena are more fully described in other connections (Chapter XIII).

**Symptomatology of Cardio-sclerosis.**—As the morphology of cardio-sclerosis is a varied one, so its symptomatology depends on the pathological factor that is clinically predominant. It is evident that it is often impossible to group these patients into one class, for the symptoms produced by the various underlying pathological changes frequently overlap. It has already been pointed out that arteriolar and capillary fibrosis may be a generalized pathological process, or may attack individually the arterial system of the heart, the kidneys, or the brain. In such types, the symptoms are predominantly cardiac, renal, or cerebral, respectively. We shall here concern ourselves only with those individuals in whom the cardiac symptoms form the chief complaint.

As with other cardiac patients, the earliest subjective symptom of cardio-sclerosis usually is dyspnœa. Its onset is gradual; it is evident at first only after severe exertion. Later, the dyspnœa occurs upon slight exertion. It is frequently associated with a sense of weight or oppression on the chest, at first transient, later, constant. Such sensations must be differentiated from other types of precordial pain and distress (Chapter XIV) from which these patients also suffer. Precordial pains may be slight and localized, or severe and radiate to the left axilla and arm, to the neck, interscapular region, or occasionally to the right arm. At first the pains occur only after exertion;

later, they may appear in attacks which rouse the patient from a sound sleep. Slight hemoptyses occurring after exertion sometimes antedate the subjective symptoms for months or even years. An enlarged congested liver, smooth and globular in outline, is another objective sign which may be found before the patient complains of dyspnœa. It is not uncommon to feel the edge of the liver several inches below the free border of the ribs. Epigastric sensitiveness to pressure is fairly frequent. It is found even when the liver is not enlarged. The cause is usually assumed to be congestion of the gastric mucous membrane; in another connection, (Chapter XIV, XXIII) I have pointed out that it is probably of neurogenic origin, the result of reflex excitation of the gastric nerves.

Pulmonary examination often reveals crepitant rales at both bases. Their presence is prognostically suggestive of subsequent attacks of pulmonary edema. The latter may be mild and occur frequently, or a very severe attack may suddenly take place after unusual exertion or excitement. Hydrothorax, usually right-sided, may also be present. Edema of the legs is slight in the beginning and is present only when the patient is up and about; when the process is far advanced, general anasarca may supervene.

The types of arrhythmias encountered in cardio-sclerosis are of some clinical significance. When decompensation is not far advanced, the pulse is regular. Extrasystoles are usually the first arrhythmia to appear; they are mostly of the ventricular variety. The next most frequent irregularity is auricular fibrillation. Heart block is usually found only in advanced cases. In isolated instances, attacks of paroxysmal tachycardia alone or alternating with auricular fibrillation, are found. In one case, for example, a man of 54 with moderate hypertension, mild cardiosclerosis and slight precordial pain, there was an attack of paroxysmal tachycardia lasting one week; thereafter the patient felt well for several months. He then developed auricular fibrillation, again accompanied by but slight precordial discomfort. This attack lasted four days. The patient was feeling quite comfortable and his condition was progressing favorably. While sitting in a chair he suddenly died. In neither attack was dyspnœa a severe or prominent symptom. In some patients the cardiac irregularities change suddenly and apparently capriciously, from one type to another, especially from extrasystoles to auricular fibrillation, or the reverse. Such variability I have found of ominous prognostic import.

There exists a small group of patients in whom digitalis does not relieve decompensation, but produces diverse arrhythmias in rapid succession; these are chiefly sinus arrhythmia and extrasystoles, either isolated or coupled. Here, too, I have found that the occurrence of such varied arrhythmias may portend a fatal outcome within a few weeks or months.

In patients with typical "senile" cardio-sclerosis—that is, those who have in addition to cardio-sclerotic changes, palpably thickened and tortuous arteries —auricular fibrillation is very common. One of my hospital services consists

of old people with cardio-vascular disease; among these there is a large proportion of fibrillators. In those who are compensated, the cardiac rate is between 70 and 80 per minute, most of the beats are effective and reach the wrist. In those who suffer from severe decompensation the cardiac activity is rapid and quite irregular, with many frustrane beats (pulse deficit). The course of the disease of the elderly cardio-sclerotic patient is essentially afebrile, unless disturbed by complications. The most frequent complication is pneumonia, chiefly of hypostatic or embolic origin.

**Prognosis in Cardio-sclerosis.**—Under this caption are included those non-rheumatic chronic cases of heart disease which show various degrees of involvement of the myocardium, and atheromatous changes of the endocardium, the coronaries, the valves and the aorta. With perhaps the exception of syphilis, the etiological factor, whatever it may be assumed to be, has long since reached the quiescent stage; the pathological process, however, apparently continues insidiously and slowly. Clinical and pathological proof of this is usually lacking, since the slow progress of the damage can scarcely show itself except by careful clinical observation of the same patient over a long period of time, or by occasional necropsies of patients in whom the disease has been watched and studied since its incipiency.

The kind and frequency of complications found in cardio-sclerosis are, in the main, different from those in rheumatic endocarditis. Prognostically, much depends upon the type of disease which dominates the clinical ensemble. For example, if the uremic element predominates, the prognosis will revolve upon that; cerebral hemorrhage or acute uremia are then apt to be the commoner ways in which the disease will terminate. If the arteriosclerotic syndrome predominates, the terminal accidents are prone to be hemiplegias or monoplegias. But the arterio-sclerotic syndrome itself can occasionally be clinically subdivided into that affecting the aorta and the coronary circulation, and that involving the myocardium; the prognosis must then be studied from these separate standpoints.

**General Prognosis.**—"Acute accidents" which may occur and which may immediately change the entire prognostic picture will be discussed later. Aside from these, the general prognosis depends on an attempt to gauge the rate of development and the extent of cardio-sclerotic damage, and the actual degree of myocardial insufficiency. The first is only derived from careful and patient inquiry into the onset of cardio-vascular symptoms, and from tangible evidence of an underlying infective process. From these the duration of the disease may be estimated. Thus, in one instance of severe myocarditis with decompensation, in a physician of 55, I was definitely able to establish the commencement of the disease as a mild nephritis following grippe infection some twenty years previously. The patient had entirely overlooked the nephritis as an etiological factor. Very slight cardiac symptoms began fifteen years ago and consisted of slight dyspnœa and occasional hemoptyses. Acute symptoms of decompensation appeared only a few months

prior to my first examinations; they usually occurred after rapid walking or after coitus. The fact that cardiac symptoms began so long ago, and the present status of widespread cardio-sclerosis as revealed upon examination, made it appear that the pathological damage must have long antedated the symptoms. The entire history bespoke a steady though very gradual continuation of the pathological mischief.

This case well illustrates the meaning conveyed, and the information to be gained, by a careful clinical history, in an attempt to roughly approximate not only the degree of cardio-vascular disease but the length of time required to reach the condition found when the patient first presents himself for examination. The degree of myocardial insufficiency is gauged not alone by physical signs, but also by its usual clinical manifestations; chiefly dyspnœa, precordial distress, and a sense of exhaustion after effort.

It will be noted that the amount of cardiac hypertrophy and of hypertension has not been emphasized, because these are more concerned with sudden heart failure and cerebral accidents than with the question of "general prognosis." Thus, specimens of extremely hypertrophied and diseased hearts have been removed from patients who lived to ripe old age, and in whom the disease had doubtless been present for many years. So, too, clinicians have for many years followed patients with severe aortic and myocardial involvement; yet during this long period there may have been no marked discomfort, and almost negligible subjective symptoms from the cardiac disease.

With our present knowledge, we cannot state why some tremendously hypertrophied hearts carry their circulatory burdens fairly well for many years, while other more nearly normal hearts suffer from early circulatory failure. I believe that the activity and the degree of progress in the pathological process play important rôles. The system may accustom itself to a gradual curtailment of its circulatory reserve, while a more acute though less widespread pathological change might be attended by quick cardiac exhaustion in a heart already working at or near its maximal energy.

In order to study myocardial insufficiency from the standpoint of prognosis, it is further necessary to evaluate roughly not only the amount of pathological damage to the cardio-vascular system, but also the amount of fairly healthy tissue that remains. For this purpose, and in addition to careful physical examination, the cardiac reserve power should be estimated in order to determine whether any "factor of safety" remains. Functional efficiency tests (Chapter XIV) may be of some value in this connection. More readily applicable and of greater aid are the facts already mentioned, which can be determined by the ordinary methods of clinical examination. These include the investigation of the result of effort and exercise upon the heart rate; the patient's subjective sensations when reacting to effort; the size of the liver, the presence of dyspnœa, edema, hydrothorax and precordial distress.

To study ideally the degree and rate of progress of the pathological damage requires a knowledge of the patient over a long period, but this opportunity is rarely vouchsafed us. We therefore, shall have to depend in the main upon the data and history furnished by the patient. Of great importance prognostically are the frequency, type and duration of attacks of decompensation, and the manner and rapidity with which they yield to proper therapy. These considerations are often glossed over or entirely disregarded; yet from them, in addition to other data, one is frequently guided correctly as to the ultimate "general prognosis." Much will depend naturally upon the individual opinion as to what is "proper therapy." If, for example, decompensation is infrequent and has been relieved, and diuresis has been promoted by diet, digitalis and some caffeine derivative (theobromin, theocin, caffeine), the "general prognosis" regarding longevity is correspondingly enhanced. On the other hand, if decompensation is frequent, severe and is refractory to treatment, the "general prognosis" is poor.

Knowledge of the prognostic features included under the term, "Acute Accidents," depends upon the fact that, in the pathology of cardio-sclerosis there is an admixture of myocardial, arterial and valvular disease in varying proportions; and it requires not only laboratory tests and detailed examination, but also clinical acumen and broad experience to decipher and properly apportion the damage to the heart, kidneys, arteries and brain. Thus aneurismal dilatations of the aorta may be confined to its first portion, the arch, or to the descending thoracic aorta, while the coronaries and myocardium may be healthy. In other patients with cardio-sclerosis, the coronaries and their branches are only slightly involved, while the brunt of the disease affects the myocardium and produces myofibrosis and hypertrophy. Conversely, coronary disease may be widespread and include subsidiary branches of the second and even third order, yet the main mass of the myocardium may remain comparatively healthy. Again, an arterio-sclerotic process may spend itself chiefly upon the circle of Willis, and produce symptoms of cerebral softening. All these instances show how necessary it is to search out the lesion which is symptomatically and pathologically predominant; for in this manner alone can a fairly scientific prognosis be grounded, and "acute accidents" foretold and possibly guarded against.

For the purpose of studying acute accidents prognostically, patients with cardio-sclerosis may be roughly grouped into those with predominant arterio-sclerotic, uremic, or cardiac symptoms. In cases with general atheroma without marked hypertension (usually found in the "senile"), the more common causes of sudden death are cerebral hemorrhage, pneumonia or pulmonary stasis. The latter produces bubbling or crepitant rales at both bases. Such signs may be present for weeks or months before an "acute accident" occurs; they help presage probable pulmonary edema or a terminal pulmonary infection.

A fairly definite hint of cardiac failure or of impending death is furnished by the presence of arrhythmias, the type of which change, apparently capriciously, hourly or daily. Thus, at various times, extrasystoles, auricular fibrillation, or tachycardia may alternate with a regular pulse. This heightened ventricular irritability is not necessarily associated with any demonstrable change in the clinical symptoms. It seems to be due to some profound change in cardiac nutrition dependent on severe sudden focal myocardial damage from coronary emboli or infarcts. The following cases will serve to illustrate the clinical picture.

Male, aged fifty-five, was suffering from mild nephritis and cardio-sclerosis with moderate hypertension. The main complaint was slight dyspnœa upon climbing stairs. He improved under therapy and felt comparatively well for one year. His pulse had always been regular until one day he complained of constant "fluttering" in his heart. Clinical examination and electrocardiograms showed a simple tachycardia; the rate was 160 per minute. His condition was accompanied by very slight dyspnœa. There was no precordial pain. The tachycardia lasted two days. For a period of three months he again felt well, his pulse remained regular. Then, suddenly, during the night he complained of feeling faint and of a very slight "uneasiness" in the chest. Again slight dyspnœa was present. Examination showed auricular fibrillation with rapid and irregular heart action; the cardiac rate was 150 per minute. He was given digitalis and later was allowed to sit out of bed. The fibrillation continued, but the patient said that he felt very much better. Despite apparent improvement and absence of pain, a diagnosis of probable coronary infarct was made, and a guarded prognosis given regarding the outcome of the attack. After four days, while the patient was sitting quietly at the table and reading, he gasped and died within two minutes.

Female, aged sixty-seven, well preserved, had been complaining of substernal pain for one year. More recently, she began to have attacks of dyspnœa and of pains lasting about one hour and radiating to the back. At such times, the pulse became irregular (auricular fibrillation); between attacks, it remained regular. Upon examination, the systolic blood pressure was 168, the diastolic, 80; there was slight pretibial edema. Fluoroscopic examination showed a somewhat dilated aortic arch. The urine contained neither casts nor albumen. The electrocardiographic tracing showed a few ventricular extrasystoles. A diagnosis of probable coronary sclerosis was made and sudden death prognosticated, mainly because of the arrhythmia which accompanied the attacks of dyspnœa. Two months after the first examination, the patient had an attack of dyspnœa and precordial pain, and died within a few minutes.

**Prognosis in Cardio-sclerosis with Coronary Disease.**—Where disease of the coronaries is assumed to be the main cause of the symptoms (Chapter XXIII) the prognosis depends on the severity of the precordial attacks and

on the extent of concomitant cardiac damage; in other words, on the state and amount of preservation of the undamaged cardio-vascular apparatus. If compensation is good, the precordial distress mild and yields to therapy, the patient may continue a fairly comfortable existence for years; in such cases, fair but somewhat guarded prognosis regarding longevity should be given. Widespread coronary disease may show itself clinically in several ways: By severe attacks of precordial distress (angina pectoris, Chapter XXIII), in any one of which the patient may die from edema of the lungs, coronary infarct, or embolus; by attacks of dyspnœa; by vomiting which has no relation to the ingestion of food and is often wrongly ascribed to indigestion and hyperacidity; or by slight febrile disturbances combined with tender areas over the precordium. Apparently the latter are due, at times at least, to focal myocardial or even to myomalaceous areas from an infective or embolic process in a secondary coronary vessel. Such a process may run its course in a few days or weeks; the heart becomes permanently affected, but the damage is not necessarily incompatible with life or with a fair amount of cardiac comfort. The prognosis in such cases depends on the severity of the infection as judged by the temperature and rigors, the duration, and especially by the frequency of the attacks. When the clinical manifestations are mild, with but occasional recurrences, the prognosis is fair, for the chances are that the myocardial damage occurring with each infection will only gradually cripple the heart, and that symptoms of myocardial insufficiency will supervene only after a long period of time. The chief danger is the possibility of embolic infarcts into some vital organ as, for example, the brain.

The main right or left coronary artery may be suddenly plugged by an embolus, or its lumen closed by an infarct (Chapter XXIII). This is usually accompanied by intense precordial distress, dyspnœa and gastric symptoms. Signs of acute cardiac failure rapidly supervene, and death usually occurs within 24 hours. Such cases have been corroborated by necropsy examinations.

Ventricular fibrillation (Chapter X) is probably another type of sudden death occurring in patients with cardio-sclerosis. As far as our knowledge goes, this arrhythmia is almost immediately fatal.[1] It seems most likely to occur in patients who receive digitalis (Chapter XX) and who, immediately preceding the presumed fibrillation attack, are in a fair state of compensation and are apparently progressing favorably. Attacks of ventricular extrasystoles (ventricular tachycardia) may occasionally presage a fatal attack of ventricular fibrillation, for we possess experimental knowledge that ventricular tachycardia can lead to ventricular fibrillation and death. Death from ventricular fibrillation may account for some necropsy reports in which neither

[1] Robinson, however, reported a case in which the patient lived several weeks after the onset of ventricular fibrillation.

the heart, brain, nor any vital organ shows an embolic cause of death, and yet the patient dies quite suddenly of advanced cardiac disease.

The degree of cardiac hypertrophy has a certain influence in increasing the probability of "acute accidents," especially after a first attack of decompensation. The cardiac reserve power is then apt to be quickly exhausted, even after restoration of compensation. Lack of cardiac reserve may evidence itself by dyspnœa, slight edema, visceral congestion, or by arrhythmias, especially extrasystoles. The usual terminal stages consist in edema of the lungs coming on in attacks, or in steadily increasing pulmonary engorgement.

Prognostically, hypertension forms an extremely important factor in the possible causation of cerebral hemorrhage. There is, however, no definite method of foretelling these apoplectic attacks. In many cases the blood pressure is high for months and years, and cerebral hemorrhage does not supervene; in others, hypertension is only moderate and hemorrhage does occur. The reason for this probably lies in the fact that this lesion depends not only on the degree of hypertension but also on the extent and type of the arterio-sclerosis. Where brain symptoms are absent, we possess no definite means of determining the extent of cerebral arteriolar disease.

Uremia is another frequent form of death in cardio-sclerosis. It is commonly reported that the diastolic pressure (Chapter XXV) is of great significance in determining the probability of uremic complications. A diastolic pressure of 130 m.m. or over is regarded as a danger signal; the higher the diastolic, the greater the chances of uremia, with all its symptoms and complications. I have not been able to definitely corroborate the assumption of a high diastolic pressure with impending uremia. Briefly, the symptoms are vomiting, nausea, headache, oliguria, dyspnœa and pallor. The special prognostic features of each of these manifestations are beyond the scope of this chapter.

To summarize, although the question of prognosis in cardio-sclerosis is complicated and entails many cardio-vascular, cardio-renal, arterio-cerebral and cardiac factors, it is possible to make a fairly accurate general prognosis in many cases. Although the time of incidence of an acute accident can be foretold in only the rarest instances, a correlation of physical data and symptomatology helps in the prediction of the probability of its occurrence.

**Fatty Heart.**—Fatty heart in the sense of an accumulation of fat pads around the heart (Chapter IV) does not of itself give rise to symptoms. It is simply one of the many local deposits common in very obese individuals. Fatty infiltration of the myocardium (Chapter IV)—a more important clinical type—is also a general phase of obesity. It is questionable whether such infiltrations alone can cause myocardial insufficiency and heart failure. It seems more likely that heart failure in obese individuals is due to the usual causes—myocarditis and coronary disease—producing cardiac symptoms perhaps more quickly because of the fatty infiltration and because the heart

muscle partakes of the flabbiness of the general skeletal musculature. The symptoms in heart failure (Chapter XIV) of this type differ in no wise from those already described. Physical signs, however, are apt to be masked because of abnormally fat chest walls and because the heart itself is encased in a fatty layer.

**The Heart in Chronic Emphysema.**—This presents a type of clinical myocarditis not infrequent, especially in men between 50 and 60. Emphysema seems to be the primary factor. The degree of myocarditis does not seem to be clinically severe. There is usually moderate cardiac enlargement; because of the emphysema or cardiac weakness or both, the heart sounds at the apex are distant. There is often a soft murmur at the apex; in advanced cases, the murmur of functional tricuspid regurgitation (Chapter XIII) is heard over the xiphoid. The arteries are soft. Hypertension is not the rule. Arterio-sclerosis is not a prominent feature. Physical signs over the lungs are the usual ones of emphysema. There is constant cyanosis of varying degrees, from slight venous stasis and suffusion to definite bluish coloration. It increases markedly upon exercise, especially upon bending the head sharply and low. When the process is not extreme, the usual complaint of the patient, in addition to shortness of breath, is a feeling of heaviness and oppression in the chest upon exercise. Severe precordial distress is rare. Edema of the legs is a late complication.

I have not been able to study these hearts postmortem, so that I cannot speak of the actual amount of myocarditis. If present, it is probably due to insufficient arterial blood supplied to the heart because of interference with the pulmonary circuit. Clinically, myocardial insufficiency seems the predominant factor, whether it be due to actual myocarditis or to right heart failure from continuous distention of these chambers.

**Therapy** is the usual one for heart failure. Special treatment should be directed to the emphysema. The results are often disappointing because the primary cause of emphysema can rarely be controlled.

## CHAPTER XVIII

## CARDIAC NEUROSIS—GENERAL CONSIDERATIONS—"WEAK" HEART—IRRITABLE HEART IN CIVIL AND MILITARY PRACTICE, WITH COMPARATIVE OBSERVATIONS—GOITROUS HEART

The role of nervous disturbances such as shock, fright, and other emotions in patients with organic disease has already been described (Chapter XIV). Other phases of cardiac neurosis such as paroxysmal tachycardia, arrhythmias and precordial pains of reflex origin are dealt with in their appropriate connections. Cardiac overstrain as a factor in initiating the symptoms found in "irritable heart" has been alluded to (Chapter XIV). One can therefore comprehend that emotional instability and cardiac overstrain, alone or combined, can in various ways be of great importance in causing cardiac symptoms in those without organic disease, especially if there already exists an underlying neurotic tendency, environmental or constitutional. The physiological control of the cardiac inhibitors and accelerators has been previously dealt with (Chapter I). It will be shown clinically that abnormal excitation of either the inhibitory (vagus) or accelerator mechanism, especially the latter, is responsible for much of the symptomatology of cardiac neurosis, and particularly of "irritable heart." Many illustrative cases of various types of cardiac neurosis are given in Cardio-vascular Clinics.

Distinct group divisions of cardiac neuroses cannot indeed always be made because symptoms and physical signs frequently overlap; I neverthless have found the following generic subdivisions of distinct clinical advantage, especially from the symptomatological standpoint. There is what may be designated as the Vaso-motor group ("weak" heart); the mildest of the neuroses; next in severity comes the Irritable Heart; and finally, the Goitrous Heart. The subject will therefore be described under these three captions.

### VASO-MOTOR NEUROSIS—"WEAK" HEART

**Clinical Symptoms.**—"Weak," "asthenic," "neurotic," "neurasthenic" hearts, X-disease (Mackenzie), are some of the terms more or less loosely applied to various ill-defined conditions in which I believe the salient feature to be an unstable state of the vaso-motor mechanism. I shall not here include a discussion of the arrhythmias which in themselves are sometimes regarded as evidence of a "weak" heart. The fact that arrhythmias of various kinds may be purely functional in origin has already been frequently empha-

sized (Chapter XI). As indicative of a "weak" heart, stress is often laid upon the presence of a soft, faint, systolic murmur over the apex, only slightly or not at all transmitted. The murmur itself is very inconstant: Some days, or at some examinations it is present; at others it is absent. Moderate hypotension (Chapter XXV) is also common in these individuals. Such findings are often interpreted as organic. Actually, however, they offer not the slightest evidence of organic cardio-vascular disease. There is never any sign of actual heart failure or of myocardial insufficiency. Subjectively the patients complain of tiring very easily; if the occasion demands it, however, they can undergo long-continued physical exertion with no sign of strain upon the circulatory system. Such physical stress, however, is often accompanied by long continued fatigue. The patients also complain of feeling faint and dizzy; sometimes indeed they actually lose consciousness. Their faces and hands easily redden or blanche, with a corresponding feeling of warmth or cold in these parts. These changes are due to vaso-motor instability, to alterations in what has come to be called the "peripheral heart," and not at all to a "weak" heart. This is further shown by the fact that these patients never suffer from edema or visceral congestion, or from any of the signs found in decompensation.

Aside from symptoms and physical signs referable to the circulation, these individuals sometimes suffer from ill-defined gastric complaints of non-organic nature, often associated with referred intercostal and precordial pains. By some, such pains are called "vaso-motor angina," a very inapt term (Chapter XXIII) in my opinion. It is because of these pains that one's attention is frequently drawn to the heart as the presumed offending organ. Nervous strain, joy, worry, excitement, and physical fatigue quickly elicit many of the symptoms I have referred to. The same factors seem occasionally to exercise an influence upon the apical murmur, which at such times becomes somewhat louder. The cause of the increased intensity of the murmur is not clear; it may consist in some disturbance of muscular tone of the ring at the mitral opening, allowing regurgitation.

The patients as a class are usually regarded as neurasthenic, because, although frequently of robust appearance, little or no physical basis for their symptoms can be found. Many have been fluoroscoped in search for some abnormality in the size and shape of the heart in order to account for the instability of the peripheral circulation. "Drop" hearts (Chapter XII) and abnormally small hearts (so-called microcardia) have been found in some of these individuals. I have studied a number of cases of "weak" hearts fluoroscopically. In some I have found the abnormal orthodiascopic types above referred to; in others, I have found the heart of normal size and contour, or with the left ventricle lying quite broad and flat upon the diaphragm. In other words there was no orthodiascopic picture that I could definitely correlate with "weak" heart. Indeed, I have made the fluoroscopic observation that some of these "weak-hearted" individuals

show particularly vigorous and strong ventricular contractions. It is therefore clearly evident that there is no constant parallelism between the vaso-motor symptoms and the muscular power of the heart.

A few brief illustrative clinical histories and findings will serve to typify some of these characteristics.

A buxom woman of 45 had been told for years that her heart was "weak." There was no history of any previous serious illness. She had had two children who died. Very soon after the death of the last child, there began a series of symptoms consisting of giddiness, nausea, and flushing or pallor of the face. These symptoms have been intensified since her menopause two years ago, so that, in addition, she often feels faint and, in fact, actually fainted several times. There is a very soft systolic murmur at the apex, the orthodiascopic tracing shows an outline slightly broader than the normal, the blood pressure and all the other physical findings are normal. There is no evidence of any organic cardio-vascular disease. This patient has been under my observation for several years. She has undergone a severe operation for appendicitis with no effect upon her circulation. When I last examined her, most of the evidence of vaso-motor disturbances had disappeared.

A vigorous woman of 40, married and the mother of two children, while abroad was suddenly called home by illness in her family. She became worrisome, and complained of feeling fatigued; her hands and face readily became cold; she complained of severe pains across the chest upon exertion. There was a faint systolic murmur at the apex; orthodiascopic examination revealed a somewhat broad left ventricle; otherwise the cardio-vascular and general examination revealed nothing abnormal. Upon being assured that her heart and other organs were normal, she soon recovered her mental poise. She began taking active exercise and now walks several miles daily without cardiac or other complaints.

A tall, narrow-chested and somewhat anemic youth of 20 complained of frequent flushes and a feeling of "heat" in the face. The lungs and cardio-vascular system were normal. The only abnormal finding was a narrow, pendulous heart. Upon being told after the examination that there was no lesion of any kind in the heart or lungs, he began leading a more normal and athletic life with rapid disappearance of the vasomotor symptoms.

I believe it is worth while emphasizing the fact that the fundamental abnormality of this entire group of patients lies in an instability of the vaso-motor mechanism, the cause of the irregular flushes, pallor, dizziness and a faintness. Nerve shock of any kind is often the culminating factor initiating the more acute symptoms. From the neurosis standpoint, most of these individuals can be classified under the term of fatigue neurosis with predominating vaso-motor symptoms. They should be sharply differentiated from the cardiac hypochondriac, a term to be applied to those with purely imaginative cardiac complaints arising chiefly from an abnormal, intense introspective tendency.

To a great extent, therapy in those with vasomotor neuroses lies in assuring the patients that there is no organic disease, and that the symptoms can be cured or at least greatly alleviated. It is a diagnostic and therapeutic error to dismiss these individuals by telling them they are "nervous," for the symptoms are real and usually beyond their control. The treatment sometimes requires patience and, always, careful individualization. The patient should never follow any form of exercise, no matter how slight, to the point of fatigue. A rest cure for a longer or shorter time may be a preliminary requirement. Patients too intent upon business must decrease the number of hours and the intensity of their work. At no time should these individuals feel hurried at their work or even at their pleasure. Rest in the reclining position, getting up late, having breakfast in bed, long rest at night, mild balneotherapy; later, graded calisthenics, or exercise in the open (walking, swimming in shallow water, even tennis) are measures which, appropriately applied and carefully selected, are of great aid to the patient in helping him regain his vaso-motor equilibrium and in finally enabling him step by step to return to his accustomed duties. In women at the menopause, endocrine disturbance doubtless plays an important, sometimes indeed the chief part in the etiology. I have tried various organic extracts alone or in combination, in order to make up the assumed deficiency in the endocrine system. In most cases, the extract of suprarenal gland in one or two grain doses three times a day, is of benefit. Organotherapy in these individuals has not gotten much beyond the stage of clinical experimentation; but I believe the latter is warranted because it is harmless when carefully prescribed although it is often disappointing in results.

When gastric disturbances are present, appropriate treatment should be advised. Among drugs I have found atropine sulphate (grains $\frac{1}{200}$ to $\frac{1}{100}$) and nitroglycerin (grain $\frac{1}{150}$ to $\frac{1}{50}$), three times a day before meals, of value. When hypotension is present, suprarenal extract is sometimes of benefit; indeed, it is often of value even when the blood pressure is normal, for it seems to stabilize the vaso-motor mechanism. Strychnine may also be helpful. If symptoms are intensified at the menopause, ovarian extract (corpus luteum) may be tried. All in all, it must be stated, that because of the type of the fundamental disorder—instability of the vasomotor mechanism—the effect of treatment is sometimes disappointing to the physician and discouraging to the patient. This is especially true in those with rather severe precordial pains (Chapter XIV).

## THE IRRITABLE HEART IN CIVIL AND MILITARY PRACTICE, WITH COMPARATIVE OBSERVATIONS

Because the "irritable heart" is so well exemplified among soldiers, I have thought it best to describe and compare it with the similar condition found among civilians, thus emphasizing the fact that the fundamental etiology

is the same. It has the further advantage of showing how the various grades may merge.

During and shortly after the Great War, medical literature abounded with various descriptions of the soldier's and recruit's heart. Indeed, so much had been written that, with but a superficial knowledge of, or interest in cardiology, the impression gained was that a new and distinct entity characteristic of warfare had been discovered, and that it is never, or but extremely rarely, encountered in civil life. This impression was heightened by the fact that similar conditions in civil life had scarcely been alluded to in the numerous reports, and that new and more or less confusing names had been given to this symptom-complex. To mention a few: The soldier's heart; effort syndrome; neurocirculatory asthenia; neurocirculatory myasthenia; disordered heart action and hyperthyroidism. From the broader viewpoint of clinical medicine; from an intensive study of the condition in cardiologic practice for many years, from the examination of hundreds of candidates before the draft board, and from observation in camp hospitals, I believe the term "irritable heart" or even "cardiac neurosis" best fits the syndrome. Either appellation applies to all types of instability and irritability of cardiac innervation both in civil and military practice in which there are assumed to be no organic cardio-vascular changes. These terms, irritable heart and cardiac neurosis, are admittedly generic. They should be so, I believe, because the condition may have different underlying etiological factors and varying clinical manifestations. For those seeking more specific appellations, perhaps vaso-motor cardiac neurosis or accelerator cardiac neurosis would more accurately define the general types.

Probably because of the early intensive cardiologic work in the late war, the fundamental relationship between the irritable "soldier's heart" and a similar condition in civil life was missed entirely or was not emphasized sufficiently. Various theories were at first propounded to explain its frequency in soldiers. For example, infection was assumed to play a fundamental role. But the numerous cases occurring in American camps among recruits who had never seen active service and who had had no infection tended to upset this theory. The fact that diminished blood alkalinity (acidosis) was found in a certain number of the cases studied abroad was also given as a tentative explanation. It seems possible that in such individuals the "acidosis" and change in the buffer salts in the blood may have been the result, not the cause, of the cardiac neurosis from circulatory disturbance and consequent interference with proper elimination of toxic products.

Intensive military training took men from civil life without regard to their previous occupations, and immediately exposed them to the stress, exertion and fatigue of camp life. I am not criticizing this procedure because the exigencies of war demand haste. Besides, it may perhaps have been wise to discover as soon as possible those recruits who were totally unfit for actual war service. Concomitant with unwonted muscular exertion, espe-

cially in those from sedentary life, there is a certain amount of nerve fatigue, perhaps best expressed by the common term "fag." Nerve fatigue, psychasthenia and neurasthenia are admittedly somewhat vague terms and are open to wide variances in meaning, but they define a tendency to quick exhaustion, inability to properly coordinate somatic and even cerebral functions, and irritability and restiveness of these functions. This nerve fatigue and instability often characterize patients with irritable hearts. In addition, there is the important emotional factor, the mental attitude of the recruit toward the war. Is he cowardly by nature? Does he fear warfare? Can the natural diffidence against war in a peaceloving people be overcome rapidly enough by instillation of the martial spirit, of military morale? Can the recruit's mind readjust itself rapidly enough to the vital necessity of risking life and limb for country? How far are such questions related to the nerve stamina of the individual? Does the recruit come of nervous stock? Has he shown any neurotic manifestations in civil life? Have venery and sex ideas played a large share in his thoughts? Questions like these undoubtedly play important roles in the mental and nerve poise of the individual, may retard or induce, considerably influence, or even cause the advent of the irritable heart.

Such are some of the important crass neurologic and psychiatric factors in camp training. These are tremendously intensified by actual warfare and combat; the nerve tension, perhaps of weeks, before actual combat; the dread of injury or death in the clash of battle; the nerve exhaustion following it; fright in all its phases—these are here the outstanding factors.

Is there any correlation between the irritable heart under camp and war conditions, and that found in general practice? I believe the two conditions are comparable, and that its infrequency in civil life is due to the relative rarity of the factors just mentioned. Besides, in general practice I believe we do not delve sufficiently into possible etiological data. We are prone to weigh the patient's story too lightly because there is no tangible, corroborative physical evidence to be seen, heard or felt. Neurotic individuals, especially those with cardiac neuroses, have frequently told me that they felt anger and excitement "in the heart," while others with gastric disturbances feel similar emotions "in the stomach." It may appear unscientific to believe such statements when no method of examination is open to us to prove or disprove them, but, measured by our own personal experiences, I am sure that there are physicians who, at some time, under special emotional stress such as fright or anger, have referred their sensations to the heart or the abdomen; and I need but recall poetic and romantic literature which abounds in examples of pangs of jealousy and rage being referred to the heart.

Illustrative cases of cardiac neurosis are given in Part II, Cardio-vascular Clinics.

The salient feature of the symptom complex of individuals with cardiac neurosis is instability of the vaso-motor or of the accelerator mechanism, or of both. I believe that all the symptoms as well as the abnormal physical

signs are attributable to these fundamental disturbances. Giddiness, flushes, pallor, dermatographia, swooning, fainting are patently due to vasomotor disturbances. They are found very frequently, for example, in many types of gastric disorder without cardiac symptoms. The tachycardia in irritable heart ranges from simple pulse acceleration to gradual or sudden, sharp increases in rate following exercise, emotion or unknown causes. The sudden, sharp increases in rate and in forcefulness of heart action are found oftenest in the irritable heart of soldiers. To use a simile derived from physiology, the threshold for the abnormal excitation varies considerably. The cause of the various types and degrees of precordial sensation, such as heaviness, pressure, pain, skin and deep sensitiveness, is still obscure because nerves of sensation have not been discovered in the heart, and because the reflex arcs involved are still matters of surmise. But the clinical fact, already alluded to, that some people in anger or fright refer the first sensation to the heart, and Head's theory of sensitized spinal segments indicate how a diseased or improperly functionating organ can cause various local referred sensations.

The abnormal physical sign of irritable heart to which most importance is attached is a thrill-like palpable systolic impulse and the thrill-like, split, or broken first sound at the apex. Soft apical murmurs that are not transmitted, slightly accentuated second pulmonic sounds, and the occasional reduplicated second sound over the pulmonary artery, the aorta or at the apex, may be dismissed because they are not characteristic of the "irritable heart" and are sufficiently common in other conditions. When present, the thrill at the apex in the irritable heart with tachycardia is systolic in time; its degree and transmission seem to depend upon the violence as well as the rapidity of heart action. A thrill, for example, is infrequent in the regular heart action of paroxysmal tachycardia, in which the heart beats at the rate of from 150 to 200 per minute. It is quite common, however, in exophthalmic goiter, even when the cardiac rate is as low as 100 to 110 per minute. I believe it is the violence of the heart action, rather than its rapidity, that accounts for the abnormal systolic thrill, for theoretically at least, it seems probable that blood, suddenly and forcefully ejected by ventricular systole, can produce impingeing countercurrents and whorls against the inner ventricular surface; to the ear and touch these are interpreted as broken, almost cog-tooth-like interruptions of the normal systolic sound and thrust. This receives some support from the observation I have frequently made that if the cardiac rate and hyperactivity can be controlled by having the patient take a long, deep breath and holding it a moment, the thrill often diminishes or disappears, almost commensurate with corresponding decrease in ventricular hyperactivity.

The close etiologic and symptomatic relationship between the irritable heart in civil and military practice are shown by the case histories with their comments. (See Cardio-vascular Clinics.) In the soldier's heart, tachycardia, especially following effort (effort syndrome), is the most common

sign; vasomotor symptoms alone are less common, while in civil practice, the vasomotor symptoms predominate. It must be remembered and emphasized that patients frequently complain of "palpitation" without tachycardia, that is, they have a subjective feeling of palpitation. In a few instances, I have been able to discover with the aid of the fluoroscope that such patients had particularly strong ventricular contractions; the left ventricle was decidedly diminished in size with each systole. This may perhaps be the physical explanation of subjective "palpitation" in some of these cases.

I have tabulated a list of the commoner symptoms, physical findings and etiological factors as I have observed them in the neurotic heart in civil practice, and have arranged each column, as far as possible, in the order of the importance of the various factors.

IRRITABLE HEART IN CIVIL PRACTICE

| SYMPTOMS | PHYSICAL FINDINGS |
|---|---|
| Abnormal precordial sensations (pain, sensitiveness, oppression). | Normal sized heart; thrill-like, split systolic impact and first sound over the mitral area. |
| Vasomotor symptoms (giddiness, flushes, dermatographia, faintness). | Systolic murmur over the lower precordium; rapid regular heart. |
| Dyspnœa (actual and subjective). | Normal rate with occasional extrasystole. |
| Extrasystoles producing subjective sensations. | Sudden changes, of heart rate. |
| Tachycardia following slight effort. | Slow heart action with powerful ventricular contraction. |
| Constant tachycardia. | |
| Sudden changes of heart rate. | |
| **ETIOLOGICAL FACTORS** | **THERAPY** |
| (Causing hyperirritability of the accelerator branch of the sympathetic nervous system:) | Reassurance. |
| Previous history of shock, fright or emotional strain. | Short rest periods. |
| Reflex excitation from gastro-intestinal canal. | Retraining, especially through graded games; bromides and luminal in the beginning or in severe cases. |
| Physical fatigue with insufficient rest. | Suprarenal gland extract (especially for vaso-motor symptoms). |
| Insufficient recuperation following acute infections. | |
| Disturbance of the organs of internal secretions (thyroid?, adrenals?). | |

Some of the headings require additional comment; others have been discussed in the description of the case histories or elsewhere.

Dyspnœa as stated may be actual, for example in patients who develop tachycardia as the result of exercise. At other times, especially in patients with vasomotor symptoms, the dyspnœa seems purely subjective, for there is no increase in the rate or depth of respiration. In rare instances tachypnea —rapid shallow breathing—results.

The size of the heart, as mapped out by ordinary methods of clinical examination, gives the impression of enlargement. This impression, however, is the usual one in almost all cases of violent, rapid heart action (Chapter XII). I have had occasion to fluoroscope many cases of tachycardia that had no organic cardiac disease. The orthodiascopic tracing revealed no characteristic change from the normal in the size or configuration of the heart. I have rarely found the drop or pendulous heart (Chapter XII), that is, a narrow organ resting lightly on the diaphragm along a small part of its under surface.

Among etiological factors of the irritable heart, a fundamental one is an "emotionally unstable, neurotic individual." (See Table.) Some of the patients give definite histories of a nervous taint in either parent, or in closely related member of the family. I believe, however, that while such taints may be common, they are not the actual exciting or impelling causes of the neurotic heart. Some great emotion such as fright, dread and shock, is usually the direct antecedent cause of the outbreak of the cardiac symptoms. Reflex excitation from the gastro-intestinal canal is the next most frequent. In other words, the cardiac nerves of hereditarily disposed individuals react more readily, more continuously and in a more exaggerated fashion to a neurogenic "insult" than do those of an individual with normal poise.

**Cause of Irritable Heart.**—Having alluded to some illustrative cases and various attendant symptoms, physical findings and etiological factors, the question arises, is there some basic underlying excitation to account for the irritable heart? Careful analysis of all the various phases of the irritable heart, especially facts elicited by careful history, lead me to the belief that it is probably fundamentally due to hyperexcitation of the sympathetic nervous system. In his classic paper on the irritable heart of soldiers, DaCosta ascribes many of the symptoms to stimulation of the sympathetic system. Recent observations of the same subject show that there exists hyperirritability of the accelerator reflex arc and probably also of the vasomotor mechanism. No diminution of the vagal tone was found, as judged by atropin experiments. Experimental observation demonstrates that fear and dread in cats, for example, are accompanied by erection of the fur, by exceedingly rapid heart action, by profuse perspiration and by staring eyes. These are all evidently due to hyperexcitation of the sympathetics. Such symptoms have their analogies in man. Fright and terror can be the immediate cause of exophthalmic goiter. Authentic causes of this type have been reported. I myself have observed at least two such undoubted cases. One of these was that of a young girl, who, while riding in a passenger elevator, saw a man killed before her eyes. On reaching home, she went to bed with rapid heart action, and within a few weeks, developed all the classical signs of exophthalmic goiter. In war recruits, the development of typical exophthalmic goiter has been occasionally observed. On the other hand, the development of rapid heart action, tremor and other signs and symptoms of the

irritable heart, without the type of goiter found in exophthalmic goiter, has been regarded by some as evidence of hyperthyroidism. Some of these individuals had soft goiters. But changes in the thyroid, especially cystic degeneration, are by no means regularly accompanied by cardiac or nervous symptoms. The subject of hyperthyroidism brings up for discussion the involved question of the endocrine glands. Are we justified in assuming hyperthyroid activity without the typical eye and thyroid signs of exophthalmic goiter? I do not believe that the present state of our knowledge allows us to go thus far. Besides, I think that the symptoms of hyperthyroidism so-called in the recruit and soldier can be understood better and more rationally on the broader and more fundamental basis of hyperirritability of the sympathetic system. It is probable that this supposition can even explain why an occasional case of irritable heart develops all the classical signs of actual exopthalmic goiter, for the endocrine glands themselves, especially those with rich nerve supply are presumably subject to varying influences of nerve excitation and nerve tone. This has been proven experimentally for the adrenals. The entire subject is further complicated by the probability that the endocrines themselves are mutually influenced by the action of hormones. This was illustrated in a case I saw at one of the war camps in which the development of a constant tachycardia was one of the prominent features. The man had gained about 50 pounds in weight within a few weeks; his breasts had taken on female characteristics; sexual desire had disappeared entirely for several months. The thyroid was not enlarged; there was no exophthalmos or tremor. Despite the rapid heart action, he looked the typical example of hypopituitarism. According to our present terminology, the case would probably be classed as dyspituitarism.

Hyperexcitation of the sympathetic system can undoubtedly produce rapid heart action, tremors and sweating; and as already stated, symptoms starting thus, may, in some instances, finally eventuate in actual exophthalmic goiter. In several polygraphic tracings of undoubted cases of exophthalmic goiter, I observed that the conduction time from auricle to ventricle (the *a-c* interval) was diminished even when tachycardia was not marked (Chapter X). This may offer some clinical corroboration of the experimental observation that one of the effects of stimulation of the sympathetic is a decrease of this interval. Only recently it was pointed out that the thyroid is subject not only to hyper- and hypo-, but also to dysfunctions, a fact which may account for some of the vagaries and apparent contradictions in the clinical syndrome of exophthalmic goiter.

The physiologic antagonism between the sympathetic and vagus systems, between the accelerators and inhibitors is of course well known. In an abnormal antagonism between these two controls, I believe that even when inhibition is normal, its power can be nullified by hyperirritable and hyperactive accelerators. In other words, the inhibitory tone, as tested by atropin, may be normal, yet its power can be nullified by hypertonic accelera-

tors. This is perhaps explicable by the physiological fact that, tested electrically, there is normally no quantitive relationship between these two systems (Chapter I). Sympathetic excitation, therefore, can apparently upset the nice balance of cardiac nerve control, with consequent lack of the normal inhibitory process.

From a review of the data I believe we may assume that the "irritable heart" is due fundamentally to sympathetic hyperexcitation, unopposed or insufficiently opposed by the inhibitory apparatus, and that therefrom arises the cardiac syndrome and vaso-motor symptoms at one extreme, and the actual goitrous heart at the other.

The fact I again wish to emphasize is that the soldier's irritable heart is no new complex, but is the same syndrome seen in civil life, intensified and multiplied by training and war conditions. Suspense and fright intensify latent and dormant symptoms and tendencies, and cause nerve instability in those temperamentally unfit, by heredity or otherwise, "to carry on."

**Therapy.**—On the continent, the original treatment of the soldier's heart had narrowed itself to a comparatively short period of rest, and then graded restraining in camp routine: Mild exercise at first, routine marches, light packs, heavy packs, etc. In this way a fair percentage again became fit for active duty. Digitalis was found to be of no value, as indeed was to be expected, for the patients did not suffer from decompensation, nor was there any evidence of organic valvular or muscular disease. At the beginning there was no attempt to attack the problem of the irritable heart from the psychologic or psychotherapeutic viewpoint. Mental and physical relaxation, especially in the form of games, and later, sports, was not then attempted. More recently, especially in the United States, games and farming have been used therapeutically. In private practice, where these matters can be individualized better, I have found that, if after a careful examination, patients could be assured that they had no "heart disease," and that all the symptoms were the result of "upset nerves," the cure was tremendously hastened. An explanation, not too scientific, but which offers a sufficient cause for the symptoms, is also of distinct benefit, especially to intelligent patients. What such patients dread most is sudden death from heart disease. With a satisfactory explanation for their symptoms they are usually relieved, sometimes permanently, of this fear. For the purpose of mental relaxation, as well as gradually increasing the amount of exercise, I suggest billiards, croquet, bowling, golfing, light farming and, in cases that are improving, rowing and even swimming in shallow water. Re-education of the tone of the cardiac nerves in order to regain stability is oftimes a slow and tedious process. Sudden sharp exercises should be avoided. The practitioner should never omit praise, even exaggerated praise, for good work done on the patient's part in overcoming his timidity to exercise, and especially in continuing exercise and play, despite the presence of such minor symptoms as moderate tachycardia and slight precordial pain.

Medicinally, I have found luminal, mixed bromids and suprarenal gland extract the best drugs. The bromids or luminal given in large doses at first, lessen mental irritability and anxiety. My usual procedure is to give luminal tablets of one and one-half grains twice daily, or one gram of the mixed bromids well diluted in water, three times a day after meals. Depending on the progress of the case, the dose is correspondingly diminished and finally stopped. In cases that are annoyed by sleeplessness, I add ten grains of chloral hydrate or eight grains of veronal every night for a few nights. I have seen no bad effects on the heart or on the individual from these procedures. The suprarenal gland I prescribe in one or two grain tablets, to be taken only when symptoms such as tachycardia or precordial distress become annoying. Paradoxical as it may seem, I prescribe this extract even when hypertension is present. Its beneficial effect seems to depend on its power to temporarily stabilize and decrease vasomotor instability by its action on the vasomotor center; it thus counteracts the cause of many of the symptoms. The effect of the drug is only temporary, hence with recurrence of symptoms, the dose should be repeated if necessary several times a day.

**To Summarize.**—The irritable heart as observed in civil practice is similar to that found in soldiers. Vasomotor symptoms are more pronounced in the former. The etiological factors are similar, but because of training and war conditions, dormant and latent neuroses crop out oftener and are more readily evoked; consequently the cardiac syndrome is more violent and lasts longer. Infection is a factor only in so far as it induces and produces nerve and muscular fatigue, and psychasthenia. There is no pathologic change in the cardio-vascular system. The fundamental cause of cardiac neurosis with its various manifestations seems due to hyperexcitation of the sympathetic nervous system, either of its accelerator or vasomotor arc, or both.

It may be here remarked that in civil life many of the febrile respiratory affections, especially when epidemic as in influenza, are followed by cardiac neuroses. These are often regarded as due to a toxic myocarditis from the infection. Such respiratory affections, however, are extremely rarely followed by actual organic changes in the heart. The fundamental cause of the cardiac neurosis seems to rest upon conditions of general nerve fatigue incidental to sharp febrile invasions with their crises, and possibly also to disturbances of cardiac functions from the toxemia.

## THE GOITROUS HEART

I shall consider only the circulatory phases of the goitrous heart, with its symptomatology, physical signs and medicinal therapy, and shall emphasize especially those features which have a bearing upon the general subject of cardiac neurosis.

From the immense literature of the subject it is clear that etiological factors of the most diverse kinds can produce exophthalmic goiter, and,

with it, the goitrous heart. It may be of interest to briefly mention some of these factors; thus: Drinking waters, pregnancy, menstruation, climacteric and other endocrine disturbances, infectious diseases, change of type of a cystic goiter, sudden emotional disturbances, thyroiditis from direct inflammatory continuity from a neighboring inflammatory focus, have all had their advocates. Although it is not known in what manner exophthalmic goiter is actually brought about, it seems proven that, once established, hyperthyroidism from overproduction of toxic products or from their abnormally rapid absorption (thyrotoxicosis) is one of the important fundamental elements in the symptomatology.

The goitrous heart may occur without the usual syndrome of exophthalmic goiter; the reverse occurs much more rarely. The characteristic finding in typical instances of the goitrous heart is violent, overforceful and rapid heart action. Similar to the physical sign already described in irritable heart (q. v.) but more pronounced and more constant in occurrence, is a rough palpable thrill not only at the apex but over the entire mitral area. This is commonly combined with an overforceful throb and impact of the ventricles against the chest wall. To a lesser degree there is a throb-like impact of the aorta and pulmonary artery against the examining hand flatly applied to the chest wall. The overaction of the great vessels and of the ventricles is often visible as well as palpable. Upon auscultation one hears a systolic thrill, or a split, cog-tooth-like first sound over the mitral area. The murmur and thrill are often transmitted to the carotids and to the axilla. The auscultatory and palpatory phenomena so resemble valvular disease that they are often mistaken for the latter; as a matter of fact, however, endocarditis in the sense of vegetations is an extremely rare autopsy finding. An occasional autopsy instance of myocarditis, apparently the result of thyrotoxicosis, has been reported. The abnormal sounds and thrills over the mitral area are apparently due to vibrations of the valves from racing intraventricular blood-currents and whorls, in combination with violent ventricular impact against the chest wall. A systolic murmur of varying intensity is also often heard over the aorta in the second right interspace.

The violence and impact of the heart action frequently give the impression of extreme enlargement; but with the exception of advanced cases with decompensation, orthodiascopic examination shows no marked difference in the size and form of the heart from the normal. Slight enlargements, however, apparently due to ventricular dilatation and dilatation of the aorta and pulmonary artery, are by no means infrequent.

While tachycardia with a regular pulse rate of from 100 to 120 is the rule, pulse rates of 100 and under occasionally occur. In a few of the latter, I found a slightly shortened conduction time (Chapter X) from auricle to ventricle. In isolated instances there are brief periods of comparative slowing followed by similar periods of rapidity of the heart action, as if the heart came under alternate cyclic control of the vagus and accelerators,

respectively. Extrasystoles occasionally occur with the goitrous heart. Of comparatively frequent occurrence is the complete irregularity of the pulse (auricular fibrillation). The arrhythmia often occurs in attacks, especially in those in whom the heart rate is already rapid. The attacks may last several hours; they seem to bear no relationship to the general neurotic or other symptoms present. In those with permanent auricular fibrillation, the clinical phenomena of heart failure are apt to play an important part in the symptomatology. That this is not always the case, however, is demonstrated by a patient, a female of 54 with exopthalmic goiter who has auricular fibrillation and who has been under my observation for several years. Despite the fibrillation she has had hardly any cardiac symptoms. She attends to household and other duties with scarcely any dyspnœa or palpitation. The only definite sign of decompensation was edema of the legs when she first came under my observation; this has gradually disappeared under digitalis therapy.

Hypertension is a frequent accompaniment of the goitrous heart; systolic blood pressures of 180 and over are sometimes encountered. It is apt to vary considerably, however, from day to day. The usual pathological changes found in the kidneys in hypertension are absent. Apparently the fundamental cause of the hypertension is hyperirritability and instability of the vasomotor mechanism.

**Dyspnœa,** often mild, is common. From the circulatory standpoint patients with goitrous hearts are usually incapable of severe exertion because the latter increases the violence and rapidity of cardiac activity. Emotional excitement is also very apt to have the same effect.

Treatment will be discussed from the circulatory standpoint alone, although it is recognized that the cardiac symptoms are but part of the clinical syndrome. When in advanced cases the usual signs and symptoms of cardiac failure are present—dyspnœa, edema, pulmonary congestion, etc., the same regime of bed rest and cardiac remedies (Chapter XX) as in cardiac failure from other causes should be employed. In those with attacks of auricular fibrillation, I have found that digitalis neither curtails their duration nor does it have any effect upon the rate or violence of the cardiac arrhythmia. In those with permanent auricular fibrillation, also, digitalis does not exert its usual beneficial effect. Some years ago, for example, I observed a patient for some weeks with auricular fibrillation and a goitrous heart. The patient was severly decompensated. Thorough digitalization had not the slightest effect upon the irregular heart action, the cardiac symptoms or the clinical course of the case. The patient became progressively worse and died. Perhaps quinidine sulphate may be of use in these cases. Except in one instance, I have had no personal experience with it in the auricular fibrillation of exophthalmic goiter. Digitalis has no effect in lessening the tachycardia and violent heart action when the pulse is regular. One may perhaps surmise that the usual cardiac remedies cannot have their accus-

tomed effects until the basic etiological factor causing the goitrous heart can be controlled. Caffein and its derivatives (Chapter XX) are sometimes of value when edema is present. The bromides given in large doses occasionally have the power of reducing the cardiac hyperactivity, thus rendering the patient more comfortable: The drug has very little power in reducing the cardiac rate. When tachycardia disturbs the patient's rest at night, veronal in 5 to 10 grain doses alone or in addition to a bromide mixture is of value. Luminal in 1 to 3 grain doses at night may also be tried. For flushes and other vasomotor symptoms, the extract of the suprarenal gland in one grain doses is beneficial even in the presence of hypertension. If the sensation of cold be a pleasant one to the patient, an icebag to the heart is occasionally of use in controlling the tachycardia.

## CHAPTER XIX

## CONGENITAL HEART DISEASE

In previous chapters (Chapters I, II, IX) the various congenital malformations and their embryological characteristics, as well as their electrocardiograms were described. The clinical findings and physical signs of the commoner and more important of these defects will now be discussed. In order to understand the special signs in individual lesions, it will be necessary to detail some of the general characteristics of congenital heart disease. Cyanosis (*morbus ceruleus*) is the most prominent obvious sign of congenital cardiac affections. It is by no means invariable; it is found especially in those in whom the defect has produced marked interference with circulation. Cyanosis, when typical, is present from birth, the infants are known as "blue babies." It is most apparent in the fingers, toes, nose and ear lobes. Cyanosis, however, may not be present until several days or months after birth; even then, it may only make its appearance when the child cries vigorously. Both in infancy and later, cyanosis is increased by any factor which embarrasses the pulmonary circulation. Various degrees and extremes of cyanosis are encountered in congenital heart disease, from a slight bluish tint evident only on exertion, to a deep violaceous, bluish purple hue of the skin. The mucous membranes are then similarly discolored: The conjunctivæ are deeply cyanotic, the buccal mucus membrane may be almost black. While the amount of cyanosis follows no definite rule regarding types of defects, it may be said that deepest cyanosis is found in pulmonary atresia and transposition of the arterial trunks (Chapter II) while next in degree is congenital pulmonary stenosis. Cyanosis is moderate in biloculated and triloculated hearts. Cyanosis is least evident or indeed may be absent in patent foramen ovale, in patent ductus arteriosus and in septal defects, except in the terminal stages or upon exertion. Polycythemia is frequently present as a concomitant of cyanosis. The red cell count may vary from 6,000,000 to as much as 9,000,000 in severe cases; the hemoglobin from 100 to 130 per cent.

The cause of cyanosis in congenital heart disease has been in dispute for many years. It has been variously explained as due to mechanical admixture of venous and arterial blood resulting from abnormal communication between arterial and venous chambers or blood vessels; to interference with the return flow of venous blood in the pulmonary and systemic circuits or to insufficient aeration in the pulmonary circuit. While any one factor cannot always be singled out as the controlling one, it seems probable that the cyanosis depends fundamentally upon insufficient aeration, whether brought about by too

much venous blood in the arterial circuit or by obstruction in the venous pulmonary circuit. Apparently also the circulation can accommodate itself to a varying degree of lack of aeration (that is, to lack of oxygen) without causing cyanosis: When oxygen deficiency reaches a certain limit, cyanosis results (Abbott).

Clubbing, especially of the fingers, less of the toes, is another prominent sign of congenital cardiac defects. The terminal phalanges are the ones particularly affected, they appear somewhat flattened, the nails become convex.

Dyspnœa is usually an early and prominent symptom in severe cases, whether cyanosis be present or not. Though dyspnœa be present in the cyanotic cases, it should be emphasized that cyanosis and dyspnœa do not necessarily go hand in hand. Cyanosis is often marked yet dyspnœa may be comparatively slight. Dyspnœa occurs in attacks, especially in the extremely cyanotic group.

Hemoptysis is frequent in those with marked cyanosis. It may vary from slightly blood-tinged sputum to massive pulmonary hemorrhages. The latter, when occurring, may be a very alarming symptom and become immediately dangerous to life.

## GENERAL PHYSICAL SIGNS OF CONGENITAL DEFECTS

The general physical signs, especially murmurs and thrills, vary not only with the type and degree of the cardiac defect and anomaly, but they also vary considerably with the state of cardiac compensation. With these limitations, and remembering that murmurs and thrills of congenital anomalies are neither characteristic nor constant (indeed, they may be absent), some of the typical murmurs will be described.

Typical congenital pulmonary stenosis and patent interventricular septum produce rough harsh systolic murmurs and thrills; in pulmonary stenosis, the latter are more prominent over the upper left interspaces; in patent interventricular septum, over the midsternum.

When present as isolated anomalies and of only moderate size, individuals with patent foramen ovale or interauricular septal defects rarely present clinical signs or diagnostic symptoms. Based upon necropsy evidences, it is apparent that patients even with large defects present no physical or clinical signs during life upon which a correct diagnosis might have been based. Cyanosis is rarely present; only occasionally are systolic or diastolic murmurs heard over the left third interspace. Orthodiascopic tracings present nothing characteristic.

## PATENT DUCTUS ARTERIOSUS (PATENT DUCTUS BOTALLI)

This congenital defect is not rare. Cardiac symptoms are not always present; but when they are, the history usually shows that the patients were

born cyanotic. The cyanosis is increased by crying, by exposure to cold, and in older children, by exertion and exercise. Upon percussion, a slight extension of cardiac dullness in the second left interspace (Gerhardt's area of dullness) can sometimes be mapped out. A rough systolic thrill is felt, and a loud, rough systolic murmur is heard most intensely over this area. The murmur is usually transmitted over the entire precordium and into the carotids. In addition, there is a loud diastolic murmur most prominent in the left upper interspaces near the sternum; the double murmur thus produced is called a "machinery murmur." The presence and intensity of the diastolic murmur apparently depend upon the amount of blood which regurgitates through the

FIG. 277.—Orthodiagram of a boy of 16 with patent ductus arteriosus. The curve of the pulmonary artery (P.A.) is extremely enlarged and flattened.

patent duct, and, in its mechanism, resembles aortic insufficiency; indeed, capillary pulsation has been described as an occasional accompaniment of this lesion. The orthodiascopic picture in typical cases of patent ductus arteriosis is that of a vigorously pulsating aortic arch, increased in size, beneath which is found an enlarged, flattened or protuberant overacting pulmonary artery. The duct itself is rarely visible because of its small size and of its frequent involvement in pericardial adhesions. This condition was found in two autopsy specimens I have seen.

The following is an illustrative case, with clinical and orthodiascopic findings. Male, aged 16, tall and spare, was born a "blue baby." He had scarlet fever when seven years old. He has never been able to play actively or to run. He frequently complains of palpitation; he has a productive

cough and has had hemoptysis. His face and extremities are cyanotic, his lips almost black, his fingers markedly clubbed. There are vigorous carotid and jugular pulsations, and a strong visible ventricular impulse; the apex is most prominent in the fifth interspace. A strong systolic pulsation is seen in the second and third left interspaces just outside the sternal border. There is a marked palpable carotid thrill felt almost up to the angle of the jaw. There is also a rough thrill most prominent in the second, third and fourth left interspaces, less marked in the second and third right interspaces. A definite (Gerhardt's) area of dullness can be mapped out by percussion. Upon auscultation, a loud systolic murmur is heard over the entire precordium, and over the chest posteriorly. The murmur is especially loud and rasping over the second and third left interspaces and over the manubrium sterni. The fluoroscope reveals a vigorously overacting aorta and pulmonary artery; the latter is extremely enlarged and flattened (Fig. 277). The electrocardiogram shows a negative S deviation in the first and second leads, a marked evidence of right ventricular preponderance.

## PATENT INTERVENTRICULAR SEPTUM

This congenital anomaly very rarely exists as an isolated defect. Cyanosis is the most prominent clinical sign; its degree depends on the size of the interventricular aperture and on the presence of other congenital defects—usually pulmonary atresia. The extremities are clubbed, there is a marked coarse systolic thrill to be felt, and a corresponding rough murmur to be heard over the entire precordium; the maximum intensity of the murmur and thrill is over the lower sternum and xiphoid and occasionally over the epigastrium. There is also vigorous epigastric pulsation. In the orthodiagram the left auricle and ventricle are hugely dilated and rounded, the heart is also enlarged to the right; because of atresia of the pulmonary artery, its pulsation may be slight or absent.

The following case illustrates a fairly well compensated patient with a patent interventricular septum.

F. M. female, aged 9, the youngest of three children was born a "blue baby." Her face was always duskier when she cried. She can now walk fairly well and can even climb stairs slowly without dyspnœa. The child is well nourished and well developed. Inspection of the neck reveals no hyperactivity of the carotids or jugulars. There is moderate cyanosis of the hands and lips, and slight clubbing of the phalanges. Inspection of the chest shows slight overaction of the apical region. Upon palpation a rough systolic thrill is felt over the mitral area; this is not transmitted to the carotids. Upon auscultation, a loud rough systolic murmur is heard chiefly over the mid-sternum and to its left; from here it is transmitted, although much less intensely, over the entire anterior surface of the chest: It is heard

faintly posteriorly. Orthodiascopic tracing revealed slightly enlarged aortic and pulmonary curves, and a somewhat hypertrophied left ventricle. Slow walking caused neither dyspnœa, tachycardia nor increase in cyanosis.

A unique instance of patent interventricular septum in a child with congenital dextrocardia also came under my observation. The history and findings are as follows:

Male, age 3½ years, was the youngest of nine children; he had measles and pertussis when two years old. The mother said the child had always been "blue" and "short of breath," but never sufficiently dyspnœic to be

FIG. 278.—Orthodiagram of a child with congenital dextrocardia and patent interventricular septum. It shows an enlarged and broadened aortic curve and enlarged "right" (see footnote p. 358) auricular curve, coalescence of enlarged and knob-like "left" auricular and ventricular curves, and absence of pulmonary prominence.

bed-ridden. The child appears well nourished. The extremities, lips, face and conjunctivæ are cyanosed; the conjunctival blood vessels are congested; there is distension of the superficial veins of the chest and abdomen. The fingers and toes are clubbed. There is a vigorous, heaving systolic impulse in the right axillary line and in the epigastrium. A strong, marked systolic thrill can be felt over the entire precordium; it is roughest and most prominent at the lower end of the sternum and in the epigastrium. Over the former there is a loud, rough systolic murmur, tailing away over the remainder of the chest, anteriorly and posteriorly. The murmur is transmitted along the carotids. There is no Gerhardt's area of dullness in the second and third right inter-

spaces. The liver and spleen are not transposed; the gastro-intestinal canal, as shown by bismuth roentgenograms, is in its normal position. Fluoroscopy of the chest reveals an enlarged and vigorously pulsating aorta. Beneath it, the usual pulsating curve representing the pulmonary artery, is absent. In its stead, there is a tremendously dilated, prominent and knob-like "left"* auricle. The "left" ventricular shadow is also very much enlarged; its area cannot be definitely separated from the superimposed auricle, although their non-synchronous pulsation can be readily determined. The "right" auricle is enlarged; beneath it, the beginning of the "right ventricular" shadow is seen deeply depressing the diaphragm during inspiration. From this finding, it is probable that the "right" ventricle is enlarged. The orthodiagram (Fig. 278) shows a greatly dilated aorta, rounded and prominent, and coalescence of the "left" auricular and "left" ventricular curves. The "right" auricular curve is also considerably enlarged. The electrocardiogram shows the typical downward deviation of the P, S, and T waves in the first lead, pathognomonic of congenital dextrocardia (Chapter IX). A diagrammatic representation of the probable circulation in this case is shown in Fig. 279.

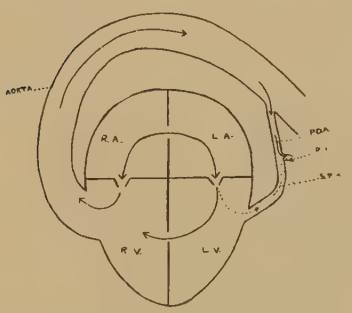


FIG. 279.—Diagram of probable cardiac circulation in the patient DL. Solid arrows = main circulation; dotted arrows = minor circulation. R.A., right auricle; R.V., right ventricle; L.A., left auricle; L.V., left ventricle; P.D.A., patent ductus arteriosus; P.C., pulmonary circulation; S.P.A., stenosed pulmonary artery.

## CONGENITAL AORTIC STENOSIS

Extreme narrowing of the aorta at the isthmus (a physiologically narrow area about one inch above the root of the aorta) is not an unusual congenital anomaly; occasionally aneurismal dilatation of the aorta finally results. Congenital aortic stenosis sometimes forms the nidus for a later streptococcus viridans infection. Thus, a young man of 21 had had "heart trouble" following a severe scarlet fever infection in childhood. For years, he had no cardiac complaints. He had occasional mild attacks of hematuria. Shortly before I saw him, he had fever and indigestion for a few days. Examination revealed loud systolic murmurs over the aortic and mitral areas. Fluoroscopic examination (Fig. 280) showed a pulsating aneurism of the first portion and arch of the aorta, and an enlarged left ventricle. The Wassermann reaction was negative. The diagnosis of probable streptococcus viridans infection affecting the aorta and mitral valves was made. The aneurism was

* To avoid confusion in the description of this case of congenital dextrocardia, "left" and "right" are used in the sense as usually applied to the normal heart in its normal position; that is, "left" will refer to the larger, and "right" to the smaller ventricle, with their corresponding auricle.

accounted for as probably due to congenital narrowing of the aortic isthmus. Later, streptococci were found in the blood. An autopsy was obtained. The aortic and mitral valves were thickly covered with typical viridans vegetations, there was a moderate congenital stricture of the aorta at the isthmus.

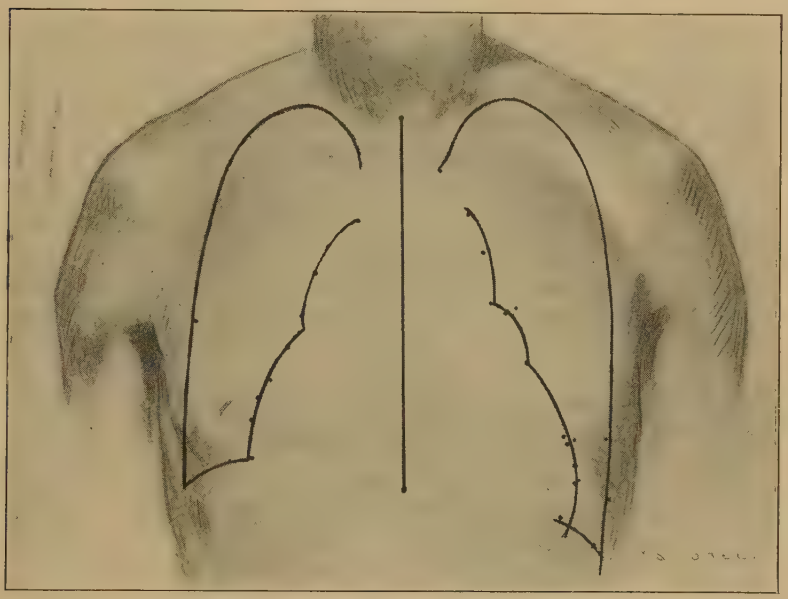

FIG. 280.—Orthodiagram of a patient with streptococcus viridans infection, a double mitral lesion and aortic aneurism. At autopsy, moderate congenital aortic stenosis was found.

## CHAPTER XX

## DIET, DRUG AND OTHER THERAPY IN CIRCULATORY DISEASE

The question of occupation for cardiacs is discussed in a succeeding chapter (Chapter XXII). The subject of special types of diet in cardio-renal disease is also subsequently described (Chapter XXI). I shall here limit myself to diet in valvular disease. Since persons with severe cardiac failure rarely have much appetite, it is as a rule safe to follow the patient's modest wishes regarding food and drink. In those with moderate heart failure the general dictum should be laid down that it is better to give food and drink frequently (about every 2 to 3 hours) instead of the usual three meals, and that the food should be bland. Special restrictions regarding salt and fluid intake are required only when edema and kidney complications are present (Chapter XXI). Bland food consists in the main of milk, cereals, gruels, clear soups, soft, boiled, coddled and poached eggs, boiled chicken, lamb chops, white meat of fish boiled or broiled, fresh and stewed fruits, thin cocoa, potatoes, creamed carrots, custards, white bread and butter, plain cake. The question of tobacco, coffee and alcoholic drinks and diet in obese cardiacs is discussed in succeeding chapters.

Intravenous 10 per cent. solution glucose injections have recently been advised in heart failure, on the theory that they assist cardiac nutrition by producing a hyperglycemia, a procedure which presumably restores the proper sugar balance in the cardiac tissue, particularly in the junctional tissues (Chapter I). The amount injected is from 200 to 300 c.c. A few favorable reports have been published. I have had no personal experience with this method.

The usual list of drugs used in cardio-vascular disease includes some which, upon careful clinical and pharmacological investigation, have been found of no or only problematical value. Only those of recognized value will be here described.

### DIGITALIS

Digitalis or foxglove was used empirically for very many years. Our knowledge of its action has recently been enhanced by the use of graphic tracings. There still exists some difference of opinion as to the method of its action. According to the most reliable clinical and experimental data, digitalis action may be divided into several components: (1) A direct effect upon the cardiac contractile power, which increases the pumping or driving force of the heart. There is good ground for the assumption that this is due

to direct action upon the cardiac musculature. (2) A marked inhibitory effect on the nervous mechanism of the heart by vagal action on the cardio-inhibitory center. (3) Effect upon the specialized junctional tissues (the atrio-ventricular conduction system). This threefold action makes it difficult to decide at times how digitalis produces the arrhythmia often found when the drug is given in full therapeutic doses. Thus, digitalis can slow the whole heart (auricles as well as ventricles) and can cause prolonged conduction time from auricle to ventricle by its primary inhibitory vagal affect. It may slow the ventricles alone in one or two ways, either by vagus inhibitory effect (thereby producing various grades of heart block (Chapter XI), or by some direct action upon the ventricular musculature. Extrasystoles occasionally induced by digitalis therapy are probably caused by the drug acting as an excitant of cardiac irritability. Auricular fibrillation can also be caused by digitalis. Unique instances of auricular standstill due to digitalis have been described. Blocked auricular beats and alternation represent other types of digitalis arrhythmias. Some of these digitalis irregularities can be made to disappear by the use of atropine subcutaneously, an evidence that they are due to abnormal vagus excitation. Some patients are hypersensitive to the neurotropic action of the drug and very soon develop cardiac irregularities. Thus, a young man of 23, with a typical rheumatic mitral regurgitant lesion, entered the hospital with the usual signs of decompensation. His pulse was regular. Several courses of digitalis were required to restore compensation. Each time, the beneficial effects of the drug were coincident with the inception of auricular fibrillation and coupled rhythm. After digitalis had been discontinued for a few days the pulse again became rhythmical. Of added interest is the fact that this patient complained of such extreme hunger and hunger pangs with the fibrillation attacks that he could not rest unless frequently fed during the night. In another connection, I have pointed out that such symptoms are probably due to vagus excitation. They are of practical importance because the advent of these peculiar symptoms is a warning that sufficient digitalis has been given. Such manifestations must be differentiated from the nausea and vomiting common in digitalis poisoning. Hunger and hunger pangs precede the usual vomiting by two or three days; hence vomiting can be avoided by discontinuing medication in those rare instances in which hunger symptoms follow the administration of digitalis.

As already indicated, the apparently contradictory effects of digitalis are explicable on the basis of its twofold action: That upon the neurogenic control of the heart, thus affecting the rhythm; and that upon the contractile power. Although these effects are usually coincident, the former may precede the latter by one or several days. In other words, arrhythmias occasionally occur before decompensation is relieved. I do not regard such early appearance of digitalis arrhythmias as any contraindication to further digitalis medication. The circulation is only rarely affected by these irregularities; the drug should be pushed in the usual fashion until the beneficial effects upon the

circulation are noted. If then the drug be stopped the arrythmias soon disappear.

**Digitalis in Auricular Fibrillation and Flutter.**—The drug exerts its greatest influence clinically on the irregular ventricular action commonly met with in auricular fibrillation; this seems due to a vagus effect upon the auriculoventricular junctional tissue whereby many of the discordant auricular impulses are blocked. As a result, ventricular action becomes steadier, slower and more regular, changes which are indicated by the pulse. In auricular flutter (Chapter XI), digitalis is employed with a double object; to benefit the cardiac failure which is usually present, and to attempt to change flutter to auricular fibrillation. When the latter is accomplished, the drug is stopped; normal rhythm is then often resumed. Many instances of the final change of flutter to sequential rhythm after the use of digitalis have been described. However, I have observed one case of auricular flutter accompanying acute rheumatic endocarditis without decompensation, in which the drug did not have this effect. The flutter appeared and disappeared several times *pari passu* with the febrile rheumatic manifestations. It finally remained absent with the subsidence of the rheumatism. One year later the pulse was still rhythmical. In other words, digitalis in large doses had no effect on the arrhythmia nor on the restoration of normal rhythm. Such negative result might have been anticipated in this instance, for the effect of digitalis in changing auricular flutter to normal rhythm probably depends chiefly upon the relief of decompensation, a marked feature in most cases of flutter.

With reference to auricular fibrillation occurring in cardiac decompensation, it remains to be added that the patient must be kept under the influence of digitalis for months or even years, in order to attain proper control of ventricular action and pulse rate. In this manner, individuals can be kept in a good or fair state of compensation for many years. Properly supervised, this long continued medication can be carried out with no harmful after-effects.

**Digitalis in Heart Block.**—If cardiac failure co-exists, complete heart block in itself is not a contraindication to the use of digitalis, for the drug cannot increase the dissociation already present. As an instance, I have reported a case of complete heart block in which the cause of the dissociation could not be ascertained during life. Digitalis was administered several times until vomiting resulted. There was no change in the dissociation, the only effect of the drug being a subjective thumping sensation in the chest. In incomplete heart block digitalis has been regarded as contraindicated because of the danger of development of complete heart block. This conversion has been reported in two cases. One came to autopsy. Lesions involving the conduction system and part of the sino-auricular node were found; but similar pathological changes in an instance of auricular fibrillation and heart block, lasting many years, have been described, and digitalis played no

role in the arrhythmia. The second case was one of severe, long-continued decompensation. Digitalis was given for several days; heart block and auricular fibrillation occurred and continued until death a few days later. These two reports do not offer sufficient evidence that digitalis alone was the cause of the block. In neither case was there clinical evidence of any ill effects from the presumed induction of complete heart block by the drug. It appears to me that the possible danger of changing an incomplete to complete block with digitalis may be averted by the judicious administration of atropine. The latter should be given in full physiological doses, administered as frequently as the digitalis, but about one half an hour before it; for example, atropine may be given before, and digitalis, after meals.

In one of my patients with complete heart block, in whom, through a nurse's inadvertence, digitalis had been continued for a long time, auricular fibrillation and coupled rhythm were produced in addition to the dissociation. These arrhythmias were not followed by any demonstrable change in the clinical condition.

**Digitalis and Alternation.**—Alternation (Chapter X)—alternate weak and strong pulse beats occurring in a rhythmical pulse—is usually indicative of a badly impaired myocardium. Thorough digitalization is indicated. Pulse alternation itself is also occasionally caused by digitalis. Because of its usual serious prognostic import when not produced by drugs, digitalis alternation might *a priori* be considered as contraindicating its further use. However, the following shows its seeming harmlessness, when alternation is produced by digitalis. A case has been reported by Mackenzie in which alternation and extrasystoles were the result of digitalis; despite this, the drug was continued and the patient showed gradual improvement. In other words, alternation so produced is apparently not dangerous and in itself does not warrant discontinuance of the drug.

**Digitalis and Extrasystoles.**—When extrasystoles are of functional origin and are not associated with cardio-vascular disease, digitalis is not indicated, although occasionally so employed on account of its power to increase vagus inhibition. When extrasystoles accompany cardiac failure, they apparently depend upon some profound nutritional disturbance of the cardiac musculature. Digitalis is then definitely indicated, for with restoration of compensation, the extrasystoles often disappear. This favorable action may directly depend upon improvement of the intracardiac circulation with consequent improvement of cardiac nutrition.

**Coupled Rhythm and Digitalis.**—Coupled rhythm is a cardiac irregularity in which every normal beat is followed by an extrasystole (Chapter X). Coupled rhythm—an occasional result of digitalis medication in auricular fibrillation—is usually regarded as a definite warning to stop the drug. In order to decide this question for myself, I kept several patients under the effects of the drug for a varying length of time after coupled rhythm had been induced. Most of the patients were elderly individuals with cardio-

sclerosis. In each case, digitalis in moderate doses was continued for several weeks after the beginning of coupling. The patients were allowed to walk about. They all felt quite comfortable, there were no ill effects. Improvement was particularly noticeable in one individual who was under treatment for a second severe break in compensation. Besides cardio-sclerosis, he had had emphysema with profuse expectoration for years. The typical gross pulse irregularity of auricular fibrillation was present. Digitalis produced coupled rhythm within five days. Medication was continued for two weeks longer, although the ventricular rate fell to 40 per minute. Cyanosis disappeared, expectoration ceased; the patient stated that he had not been so free from cough and expectoration for years. Digitalis was then discontinued; coupled rhythm and improvement remained; the patient passed from observation a few weeks later.

**Digitalis Vomiting. Administration of Atropin with Digitalis.**—Digitalis vomiting has been ascribed to irritation of the gastric mucosa, but this scarcely explains the marked variations in susceptibility to the emetic action of the drug. Some patients vomit almost immediately, others only after massive doses have been given. A very suggestive explanation is found in experiments in cats in whom the alimentary tract was removed; none the less, all the accompanying manifestations of emesis occurred when digitalis bodies were injected intravenously. These experiments strengthen the belief that digitalis vomiting is probably due to central nerve action and not to peripheral irritation. This fact is of practical clinical importance because, in susceptible patients, small initial doses of digitalis may be advantageously combined with atropine sulphate in doses of $\frac{1}{150}$ of a grain; subsequently the digitalis dosage may be increased gradually to the usual maximum. In this manner I have succeeded in administering digitalis to some patients in whom even small doses ordinarily produced vomiting. Details of the combined administration of digitalis and atropine are given in the following illustrative cases:

Male, age 60, suffered from chronic bronchitis, dyspnœa and asthmatic attacks for many years. The patient was cyanotic; physical signs of chronic bronchitis, emphysema and myocarditis were present; the pulse was completely irregular (auricular fibrillation). The tincture of digitalis and Karrell diet (q. v.) were given. At the end of one week he was very much improved; digitalis was discontinued because of vomiting. After two weeks the drug was again given with the same result. On the third administration it was combined with atropine sulphate, grain $\frac{1}{100}$, later, grain $\frac{1}{150}$, given subcutaneously. In this manner digitalis was administered continuously for several weeks without inducing vomiting and with excellent clinical results.

Female, age 27 years, had auricular fibrillation, a double mitral lesion and a decompensated heart. Fifteen minims of tincture of digitalis, three times a day were prescribed. Upon three occasions, after three days administration, medication was discontinued because of nausea and headache. On two subsequent occasions digitalis was combined with atropine sulphate. Once, one-

fiftieth of a grain was given subcutaneously when nausea was already present, and continued in doses of $\frac{1}{150}$ grain three times a day. Upon the second occasion, atropine and digitalis were given uninterruptedly for two weeks without nausea. Decompensation was relieved.

Female, age twenty-six, had a rheumatic mitral regurgitant lesion. There were cardiac pains and broken compensation for several months. Tincture of digitalis, one drachm daily in divided doses, was given. Usually after a few days' medication was discontinued because of vomiting and headache, and the decompensation which had been temporarily relieved, soon recurred. Caffeine, digipuratum and tincture of strophanthus were substituted for the tincture of digitalis without effect. Finally the latter was combined with atropine sulphate, grain $\frac{1}{150}$, given internally, three times a day. This was continued in two weekly periods, with interruptions, for many months, and the patient showed improvement. Her appetite remained good. She finally decompensated again and died.

**Digitalis and the Kidneys.**—It is now definitely known that the diuretic action of digitalis in clinical conditions in man is due directly to the relief of heart failure and to an improvement of the general circulation in which the kidneys take part. Digitalis does not cause increased diuresis in normal individuals nor in circulatory failure without edema. Furthermore, it causes no increased urine output in kidney disease, with or without edema, in which the heart itself is not implicated. Since, however, myocarditis is often associated with kidney disease (Chapter XVII), the beneficial effects of digitalis as a diuretic can be readily appreciated.

The preparation and dosage of digitalis have long been matters of dispute. I prefer the tincture, or an infusion freshly prepared from potent leaves. A good tincture kept in a properly stoppered bottle will retain its strength for several months. The objection to the infusion is that it must be freshly prepared, requires large dosage, and is more unpalatable than the tincture. The tincture and infusion are therapeutically alike. I have also found digipuratum or as they are now called, digitan tablets an excellent and reliable preparation; it is standardized in frog heart units. One tablet of one and one half grains each is equivalent to fifteen minims of the tincture. It sometimes seems more efficacious than the tincture, especially when administered in chronic cardiac disease. Another excellent preparation is the powdered leaf given in two to four grain doses four times daily. Recently, Hatcher has divided digitalis into a chloroform-soluble and a non-chloroform-soluble fraction. The latter is thick, tarry and much less active than the former. The chloroform-soluble fraction (now also called the purified tincture) has shown very active digitalis properties when used experimentally in animals; it has also shown quick absorbability and powerful digitalis action when used in man. The dose and preparation, however, have not yet been standardized.

Variability in absorption is no doubt a large factor in variability of the action of digitalis when given by mouth. By the effect of digitalis on the

T wave (flattening or inversion of the T wave in Leads I and III, Chapter IX) it has been shown that beginning digitalis action may occur even within a few hours of the administration of the drug. Digitalis dosage should be regulated by the degree of cardiac failure and by the type of cardiac disease. In general, very little if any good is accomplished by beginning with doses very much below the average, partly because small doses pass so readily out of the system. The usual total amount of the tincture required for a full therapeutic effect varies from one to one and a half ounces taken over a period of about one week. The average daily amount is one drachm given in fifteen or twenty minim doses. It is best administered undiluted because it has been found that the admixture of water may interfere with absorption. If urgency demands it or if one wishes to digitalize more rapidly, very much larger initial amounts can be safely given. Eggleston, for example, has given initial doses of several drachms of the tincture with great benefit and with no untoward symptoms except occasional nausea and vomiting. In urgent cases I have also often administered the tincture in drachm doses four or five times daily; no ill effects were observed. The usual dose of the infusion is from one half to one ounce given three or four times daily. This amount can likewise be considerably increased if quick effects are desired.

The therapeutic effect of digitalis in patients with rhythmical heart action becomes evident rather by improvement in the signs of decompensation (lessened dyspnœa and edema, increased urine output) than by any marked slowing of the pulse. In auricular fibrillation with a completely irregular and rapid pulse, the therapeutic action is shown in addition, by the elimination of the irregular and weaker ventricular contractions, the frustrane ventricular activity which produces no pulse beats (so-called pulse deficit). In consequence, ventricular rhythm becomes steadier and slower, and the pulse tends to become correspondingly regular. Some observers advise discontinuance of the drug or decrease of its dosage when this result has been achieved. This advice should be followed only if the drug has produced some degree of intoxication such as nausea or vomiting, if decompensation has been relieved, or if the usual amount of digitalis (about one ounce of the tincture) had already been taken; for it occasionally happens that the typical discordant arrhythmia of auricular fibrillation comes under sudden control after a few doses, while signs of heart failure continue or quickly recur if digitalis is then stopped. In other words, with the exceptions noted, the administration of the drug should be continued until decompensation is restored.

In decompensated cases with a rhythmical pulse and moderate tachycardia, the latter is sometimes reduced to normal rapidity with gradual restoration of compensation by the administration of digitalis. This is occasionally brought about by the inception of such digitalis arrhythmias as sinus slowing, sino-auricular block and blocked auricular beats. As already indicated, a moderately slow pulse rate is in itself no contraindica-

tion to the further administration of the drug, although the type of digitalis arrhythmia under discussion is apt to be coincident with the full therapeutic effect.

Older patients with cardio-sclerosis and decompensation usually require larger and longer-continued digitalis doses than younger individuals with cardiac failure. The former are often comfortable with ventricular rates between 45 and 50. Such elderly individuals should be kept under full digital effects by giving them several courses of the medication over a series of weeks. This is best accomplished by giving one ounce of the tincture (about one drachm daily) for the first week; it is then discontinued for the following week. In the third week about one half ounce of the tincture will be required. It should then be again discontinued for a week. In the succeeding weeks somewhat smaller doses are adequate. In this manner, the heart can be safely saturated with digitalis and the patient kept under its influence for a long time.

Much has been written about the cumulative effect of digitalis and the dangers therefrom. Variability in gastric absorption may be one of its causes. Unless the usual mild toxic symptoms are meant—headache, nausea, arrhythmias—digitalis cumulation is an infrequent phenomenon, and its dangers have undoubtedly been exaggerated. Perhaps the "cumulative effect" is best explained by our present knowledge of the persistence of digitalis action, described below. Digitalis has been administered in innumerable instances for weeks and months, and even years, with no dangerous effects. In only one instance did I observe symptoms which might be interpreted as due to an actual cumulative effect. A woman of 40 with a double mitral lesion, orthopnœa, anasarca, cyanosis and auricular fibrillation entered the hospital *in extremis*. Infusion of digitalis in one half ounce doses was given every four hours. After three ounces had been taken, the patient's condition improved. Vomiting, a drop of ventricular rate to 60, and cold face and extremities suddenly developed. Hypodermic injections of strychnine and caffein had no effect upon this condition. Despite these apparently alarming symptoms, the patient felt quite comfortable, orthopnœa temporarily disappeared, cyanosis decreased, and the urinary output markedly increased. The patient died two days later from cardiac failure.

As shown electrocardiographically by the action on the T wave the persistence of the action of digitalis in a thoroughly digitalized subject lasts two weeks or more after its discontinuance. This persistent action seems due to fixation of a small amount of the drug in the cardiac tissue (Eggleston). It substantiates the clinical observation that, after thorough digitalization, the effects of the drug can be maintained by giving much smaller doses.

It is conceivable that there is one possible danger in administering digitalis in unnecessarily large doses over long periods; that is, the production of ventricular fibrillation (Chapter IX), an arrhythmia which, so far as we now know is universally fatal. We possess neither experimental nor definite clini-

cal grounds for the assumption that ventricular fibrillation may be caused by digitalis. I predicate it upon the power of digitalis to incite all types of cardiac irregularities, and upon the occasional clinical observation that patients who are apparently improving under full digitalis dosage sometimes die quite suddenly, with no sufficient cause at autopsy for the sudden fatal termination. Some of these deaths are assumed to be due to ventricular fibrillation.

**The Use of Digitalis in Valvular Lesions.**—Dogmatic distinctions were formerly made regarding the various types of valvular lesions in which digitalis was indicated or contraindicated. Abundant clinical experience has shown that, although not followed by equally good results, digitalis may be given in all forms of cardiac failure, regardless of the valve affected. In rhythmically beating hearts, digitalis is not usually followed by the same rapidly beneficial effects as in auricular fibrillation. Its chief value in the former lies in the occasional reduction of the cardiac rate and particularly in strengthening ventricular contractions. In aortic lesions with decompensation and left ventricular hypertrophy, digitalis is not as beneficial as in mitral lesions. This may depend upon several factors: In aortic lesions there is less systemic congestion and edema, except in the final stages of decompensation; the cardiac rate is very susceptible to neurogenic and other influences which cause tachycardia (Chapter XV); finally, digitalis may not exert its full action upon the fibers of an extremely hypertrophied muscle. In mitral lesions, tachycardia is not as common; congestion and edema more common, conditions more readily influenced by digitalis through increase of the cardiac contractile power.

The value of digitalis in cardio-sclerosis with hypertension and coronary disease has often been questioned; indeed, for many years, hypertension was regarded by many as a distinct contraindication to digitalis. Careful clinical study has shown, however, that digitalis in therapeutic doses does not regularly raise the blood pressure; when occasionally increased, the increase bears no relation to the time of administration or dosage of the drug. I have treated patients with hypertensive disease, many of whom had a systolic blood pressure of over 200 m.m. of mercury, and I have never observed any pressure increment which could in any way be correlated with digitalis administration. In fact, in those in whom cyanosis and dyspnœa were the chief manifestations, and in whom digitalis gave relief, the blood pressure was often lowered. This may be because cyanosis itself acts as an excitant to the vaso-constrictor center and thus raises blood pressure. This disturbing factor is eliminated by the beneficial effect of digitalis upon the general circulation.

Coronary disease had previously been regarded as a contraindication to the use of digitalis, upon the assumption that it caused coronary spasm. There is no actual experimental evidence of this. Clinically, I regard some types of coronary disease with precordial pains as especially amenable to digitalis therapy because of the necessity of increasing the intra-cardiac

circulation. While often disappointed in its results because of the probable occluded lumen of the coronaries (Chapter XXIII), I have seen no harm from its use.

Another important consideration in digitalis therapy is the question of its use in endocarditis and pericarditis during the acute stages. When the heart rate is increased as the result of these acute inflammatory processes, digitalis has no effect in diminishing the pulse rate. This is also true of other febrile diseases, such as pneumonia, grippe, etc. Hence the use of digitalis for the persistent tachycardia of inflammatory conditions is very apt to be followed by disappointing results.

In this connection I wish to point out one reason for the futility of digitalis in chronic pericarditis with dense and extensive adhesions. The latter act mechanically like a vise, hindering normal systole and diastole. I observed one instance in an adult who had rheumatic arthritis and irregular temperature for several weeks. Auricular fibrillation was present. The obscure physical signs—a scarcely audible or palpable apex beat—pointed to myocarditis as the probable cardiac lesion. A roentgenogram of the chest showed a somewhat enlarged cardiac shadow. Digitalis was given for several weeks with no effect upon the slowly increasing heart failure or upon the fibrillation. At necropsy the valves were normal, the myocardium not extensively diseased, there was a tightly adherent pericardium that encircled and fixed the entire heart. In another instance, a young adult male, dyspnœic and cyanotic, with polyserositis of unknown origin, X-ray plates showed definite, sharply defined calcareous plaques in the pericardium. Auricular fibrillation with a fairly regular and slow ventricular rate was present. The cardiac impulse and sounds were weak. Here, also, it seemed probable that the vise-like action of an adherent and thickened pericardium was an important etiological factor in the cardiac failure and cyanosis. Digitalis had no influence upon the symptoms or arrhythmia.

Speaking from a broad clinical aspect, in decompensated valvular and cardio-vascular disease in which infection is either quiescent or non-existent, I have found the following classification of value from the viewpoint of the probable effect of digitalis medication:

1. Mitral lesions with rhythmic hearts: Improvement slow unless in the presence of marked visceral congestion. Rest is apparently the largest factor in the improvement.

2. Mitral lesions with auricular fibrillation: Unless decompensation is extreme or long continued, improvement is very rapid under digitalis. The irregular cardiac activity is quickly controlled, and, with it, decompensation is usually promptly relieved.

3. Aortic lesions with slight or moderate ventricular hypertrophy: The drug is not of much value, rest is the important factor.

4. Aortic lesions with extreme ventricular hypertrophy: Reaction to digitalis not good, possibly because there is not sufficient healthy cardiac

muscle upon which the drug can act. Even if auricular fibrillation be present, digitalis is not apt to be followed by beneficial results.

5. Cardio-sclerosis with decompensation: Those with cardiac failure and edema, with or without hypertension, are apt to react well to Karrell diet, (q. v.) and digitalis and theobromine sodium salicylate administered on alternate days. In cardio-sclerosis and hypertension with uremia and no edema, digitalis has very little effect in the relief of decompensation.

Summing up the effect of digitalis medication, the following conclusions seem warranted:

1. The best single criterion of the amount and duration of digitalis administration is its clinical effect.

2. The beneficial effect of digitalis in rhythmically beating hearts depends chiefly upon producing increased contractile power of the cardiac musculature.

3. The gradual production of arrhythmias is usually coincident with full clinical effects.

4. If decompensation demands the continuance of digitalis, the rapid onset of arrhythmias does not contraindicate its further use.

5. Atropine sulphate, grains $\frac{1}{150}$ to $\frac{1}{100}$, three times a day, given internally or subcutaneously at the beginning of medication, occasionally prevents nausea and vomiting in susceptible individuals.

6. The hypertension of cardio-vascular disease does not contraindicate the use of digitalis.

7. To derive clinical benefits quickly, digitalis should be given in full therapeutic doses. In urgent cases, very large initial doses can be given.

8. When digitalis acts promptly in small doses, the effect is likely to be temporary. For permanent improvement, thorough digitalization is required.

9. Digitalis occasionally produces epigastric sensitiveness and hunger pangs which appear to be due to heightened vagal tone. These symptoms precede vomiting by one or two days, and indicate a full therapeutic digitalis effect.

10. The effect of digitalis on the T-wave of the electrocardiogram has been described in another connection (Chapter IX). It should be emphasized here that the effect is simply electrocardiographic evidence of action of the drug upon the cardiac musculature and is not by any means necessarily concomitant with the full clinical effect of the drug. The inverted or flattened wave produced by digitalis does not invalidate any of the above conclusions.

**Tincture strophanthus.**—This possesses no advantages over the tincture of digitalis. Its disadvantage is that it varies considerably in strength, as well as in absorbability; hence it may occasionally produce a sudden unexpected toxic effect, even when given in ordinary therapeutic doses.

**Crystalline Strophanthin** is a most powerful digitalis glucoside. The best preparation is that put up in glass ampoules containing 1 c.c. of a 1 per cent. solution. The dose is from 10 to 15 minims; this should be slowly injected,

preferably intravenously. It is of remarkable efficacy, particularly in the acute heart failure of auricular fibrillation. Within a very short time, sometimes even after one hour, the injection may be followed by slower and more regular ventricular activity, with a correspondingly good effect upon dyspnœa and cyanosis; frustrane beats may quickly disappear, edema show some evidence of clearing up, and diuresis increase. If the urgency of the case demand it, the injection may be repeated at the end of 24 hours. In other words, these injections in a remarkably short time can produce the effect which only occurs after several days of the usual method of digitalis administration. On the other hand, the effect is more evanescent; hence, in the decompensation of cardiac disease, it is necessary to follow up these injections by digitalis given in the usual manner. Strophanthin is also indicated in acute cardiac failure from cardio-renal disease, especially if pulmonary edema is present. There is one caution necessary respecting strophanthin; namely, it is contraindicated, or should be used very sparingly, if the patient is already digitalized, otherwise very serious toxic symptoms may immediately follow. For example, in a patient with mitral regurgitation, extreme decompensation and rhythmical pulse, who had already been thoroughly digitalized, this precaution was not heeded by the physician in attendance; an injection of 1 c.c. of strophanthin was immediately followed by auricular fibrillation, a condition which lasted until death a few days later; strophanthin undoubtedly hastened the fatal termination.

**Tincture of Squills.**—In doses of 15 to 30 minims this drug has an action on the circulation similar to digitalis. Like the latter, it may produce sinus arrhythmia and heart block, apparently by increased vagal inhibition. Given in large doses, it has an effect upon auricular fibrillation and upon the T-wave similar to digitalis. It possesses no advantages over digitalis, and is, in addition, irregular and uncertain in action. Squills is often prescribed in pill form as a diuretic.

**Apocynum.**—Apocynum or Canadian hemp, given in fluid or solid extract, has been studied to a slight extent; it has no regular or marked effect in improving the circulation.

**Quinidine.**—Quinidine is a quinine derivative, a stereo-isomer of quinine. Quinidine or its more soluble salt, quinidine sulphate had found its way in medicine because there had been occasional sporadic clinical observations that quinine had the power of restoring the normal rhythm in cases of auricular fibrillation (Wenckeback). Frey, in 1918, was the first to study the subject systematically. More recent reports (Levy, Lewis and others) deal with quinidine from the experimental and clinical standpoints with the aid of the electrocardiograph. As a result, the following conclusions of the effects of quinidine sulphate seem warranted. One effect is directly on the auricular musculature by which it lowers the sino-auricular rate, depresses and prolongs the conduction time from auricle to ventricle, and lengthens the refractory period of the auricular musculature. The second effect is a neurogenic one

upon the vagus; the drug paralyzes, releases or decreases vagus control. It thus appears that in this regard digitalis and quinidine have opposite effects, for, as already noted, digitalis increases vagus inhibition.

Auricular fibrillation is now regarded as basically due to blocking of a continuous "circus" wave of excitation (Chapter XI), the normal method of the spread of impulses through the auricles. Although the circus wave continues on in its circuitous path, its blocking depends upon differences and defects in conductivity, irritability and refractory periods (Chapter III) of various portions of the auricular musculature: The consequence is what we know clinically as auricular fibrillation (Chapter XXI). When quinidine is administered in auricular fibrillation, its most important effect in restoring the normal rhythm is its power to lengthen the refractory period in the auricles. The mechanism of auricular fibrillation may perhaps be aptly compared to a circuit whose path has been irregularly interrupted by hurdles of all kinds and sizes; the hurdles represent the blocking and breaking up of a single wave into fibrillatory waves (auricular fibrillation). Quinidine removes these by allowing a sufficient interval for the refractory phase to pass off, so that the circus wave may again re-enter and pass on smoothly across the gaps.

It has been shown by electrocardiograms with leads taken directly from the chest (that is, electrodes fastened directly over the precordium instead of to the extremities in the usual way) that one of the first effects of quinidine is to partially regularize the fibrillatory waves; the latter have a tendency to become more regular and slow, somewhat similar to what is seen in impure flutter (Figs. 159, 161), indeed actual flutter may result. Before normal rhythm is restored by quinidine in auricular fibrillation the first clinical evidence of its action is usually a preliminary phase of increase of the ventricular rate, an increase of tachycardia; in fact tachycardia sometimes persists even after the normal rhythm has been restored. This effect of quinidine seems due to release of vagus inhibition. Occasionally the tachycardia is violent, with the natural result that cardiac output is decreased and dyspnœa results. This, and the fact that quinidine can cause gastro-intestinal disturbances—nausea, vomiting and diarrhœa—show that the drug has its dangers. Another occasional occurrence coincident with the restoration of normal rhythm is cerebral embolism from auricular thrombosis due to sudden slowing of the circulation in the auricle. Finally, quinidine exceptionally produces a shower of ventricular extrasystoles—ventricular tachycardia. These do not always occur at the beginning of quinidine medication nor is there any clinical method of detecting them except by electrocardiograms. Since we know from animal experiments that ventricular tachycardia is sometimes a forerunner of death from ventricular fibrillation (Chapters X, XI), and since in clinical auricular fibrillation, we are already dealing with a disordered rhythm and a diseased heart, caution is demanded in the use of quinidine. Indeed, I feel that the more extended and safe use of

quinidine in general medicine, and not in hospitals alone to which its use is now practically limited depends upon some method of knowing in advance which patients are likely to react by ventricular tachycardia, a dangerous or possibly even fatal arrhythmia.

Patients present favorable and unfavorable drug susceptibility (idiosyncrasy) to quinidine as indeed they do to almost all powerful drugs. Thus, with some, normal rhythm is restored after a comparatively small dose. Others again show the bad effects—especially, significant increase of tachycardia and dyspnœa—after small dosage. As a rule, normal rhythm is restored on the day succeeding the quinidine therapy. Sometimes the rhythm remains regular for a day or two, and then fibrillation recurs. In favorable cases, the normal rhythm remains permanently; "permanent" is here used in a limited sense of months, since the drug has not been employed for a sufficient length of time to use "permanent" in its true sense. Fairly frequently, the normal rhythm is disturbed by occasional extrasystoles; electrocardiograms show that they are usually auricular (Chapter X) in type, an evidence of the selective action of quinidine on the auricular musculature.

**Clinical Observation and Dosage** (see also Cardio-vascular Clinics).—Before quinidine sulphate is used, the first all-important step is to properly digitalize the patient where decompensation is present and digitalis had not yet been employed. When the full effects of digitalis have been induced, and not until then, quinidine sulphate administration may be begun. I have tabulated the results of quinidine sulphate in some 40 cases of auricular fibrillation in hospital and consultation practice. Of these about one half were old individuals with marked cardio-vascular and cardio-renal changes, some with, others without hypertension (q. v. Cardio-vascular Clinics). Many were decompensated. The net result of quinidine administration was restoration of the normal rhythm for a day or two in a few of these old people. A few complained of nausea, one or two vomited. Several exhibited an increase of tachycardia but none to an alarming extent. Some of the patients had two or three courses of quinidine but the results were the same. I had not expected permanent restoration of normal rhythm in these advanced cases of cardio-sclerotic disease, for it was my clinical impression that quinidine was of most value in younger individuals with valvular disease and without far advanced degeneration of the heart muscle.

Quinidine is administered in various doses and intervals. My own routine consists in giving six 5 grain capsules as follows: The first capsule is given early in the morning. If at the afternoon visit, there is no contraindication two other capsules are given an hour apart. The patient is again visited the same evening. If then normal rhythm has not been restored or if there are no untoward symptoms—no vomiting, diarrhœa, undue tachycardia or dyspnœa, 5 grain doses in hourly intervals is repeated for three more doses. Thus 30 grains are given. I have found this method and dosage satisfactory, and up to the present, have observed only an occasional alarming effect from

the drug thus administered. The method seems safe because any important drug idiosyncrasy (except possibly a shower of ventricular extrasystoles) will show itself with the first 5 or 15 grains, a dose which seems well within conservative limits.

I have also tabulated my results in eight patients whom I saw in consultation but who remained at home under the care and supervision of their own family physician. I felt that the risk was justified, for unless quinidine is used more widely by the general practitioner, it can have only a limited use in medicine. Besides, it is often impossible in private practice to demand hospitalization for a few days in order to test the efficacy of a drug; indeed, the request alone is apt to frighten off many patients. The family attendants in the eight cases to which I refer were physicians of average ability, none of whom had any special knowledge of cardiology. When necessary, first digitalis and then quinidine were given according to the method just outlined. The physician was told to visit the patient after the first and after the third dose (*i.e.*, after 5 and again after 15 grains had been taken). The special possible dangers—significant increase of tachycardia, dyspnœa, vomiting, nausea and diarrhœa—were emphasized. An exceptional case of cerebral embolism with regularization of the rhythm may naturally occur both in private and in hospital practice; hence this in itself is no contraindication to its use in private practice. Not more than 30 grains was administered.

Of the eight cases in private practice thus supervised and treated, most were middle aged: Six had mitral disease; two had cardio-sclerosis. The rhythm was permanently restored in three, temporarily in two; it had no effect on the remaining three. In none did the quinidine have any serious untoward effect. In one, two courses of quinidine were necessary in order to restore the normal rhythm permanently.

Where normal rhythm has been restored and occasional extrasystoles occur, it offers definite clinical evidence that the quinidine is beginning to lose its effect. In such individuals, quinidization should be continued. There is here no established method of dosage or procedure. Depending upon the number of extrasystoles, my present practice is to give the drug in 5 grain doses two to four times weekly.

Since many patients with auricular fibrillation when properly digitalized, are comfortable, sometimes for years, it is a fair question to ask why quinidine should be given at all. First, the patients lose the annoying sense of palpitation, almost always present in fibrillators. Furthermore, it is a clinical fact that if the normal rhythm be restored, most patients have much more cardiac reserve and can be more active without becoming uncomfortable. Thus, in one of my cases, tachycardia and dyspnœa continued despite rest and several courses of large doses of digitalis. The patient was practically an invalid and was scarcely out of the house for months. When the rhythm became regular, the patient was soon enabled to take up her old activities,

including household duties, climbing stairs, walking in the open, etc. The rhythm has now been normal for several months.

As a summary of indications and contraindications to quinidine, the following seem permissbile in our present knowledge.

Patients with comparatively recently established auricular fibrillation and with valvular disease in whom there is no or but moderate cardiac enlargement react best to quinidine.

Patients with advanced hypertrophy from valvular or cardio-sclerotic disease do not react favorably. The normal mechanism is rarely restored.

Patients who complain early of cardiac discomfort, and in whom none-theless, quinidine is pushed, are especially prone to develop more serious complaints—embolic infarcts, more severe tachycardia, showers of extrasystoles (R. I. Levy).

Patients whose decompensation has not been relieved by properly directed therapy because of their cardiac muscle exhaustion, are prone to develop mural thrombi in the auricles under quinidine, and hence also embolic infarcts in various organs (R. I. Levy).

Signs of peripheral venous thrombosis should be looked for in the veins of the lower limbs of individuals to whom quinidine is to be given. Such thromboses indicate a tendency to intravascular clotting, hence the drug is contraindicated in such patients.

There are three chief dangers to the use of quinidine. (1) Auricular thrombosis with cessation of fibrillation and consequent slowing of the blood current. Some clinical indications of patients in whom this is likely to occur have been given.

2. Significant tachycardia with precordial discomforts. Quinidine regularly produces a preliminary, though slight tachycardia. When tachycardia is marked it contraindicates further use of the drug.

3. Ventricular extrasystoles (premature contractions) during the time that the auricles are still fibrillating. Unfortunately, there is no clinical method of detecting these; they require graphic control for their detection. The danger lies not in isolated extrasystoles, but because they come in runs and showers (ventricular tachycardia) and thus possibly presage ventricular fibrillation and death.

**Caffein and Its Derivatives.**—Caffein produces vasoconstriction by its action upon the vasomotor center; it also has a slight effect upon the heart itself. Its main therapeutic value rest upon action on the kidneys. Since cardio-vascular disease includes the kidneys, a discussion of caffein and its derivatives is important.

Caffein acts primarily as a diuretic, thereby increasing the output of urine. The manner of its action is still in dispute; some ascribe it to an irritating effect upon the renal epithelium, others to an effect upon the renal circulation. Caffein increases principally the watery constituent of the

urine; there is also an increased output of solids, especially of sodium chloride, and to a lesser degree, of the nitrogenous constituents.

The best method of caffein administration is in the form of caffein sodium salicylate, a readily soluble salt. A 20 per cent. solution of this salt in distilled water lends itself admirably to hypodermic administration; the dose is from 15 to 30 minims.

**Theobromine Sodium Salicylate.**—This salt produces more marked diuresis than caffein and is in general preferable to the latter. Another advantage is that it has no effect upon the vasomotor center, and hence does not produce blood pressure changes. The drug is administered in 7 to 15 grain (0.5 to 1 gm.) doses three or four times daily, well diluted in water. Because of its unpleasant taste, it can be given in wafers. If the medication upsets the stomach, it may be given in smaller doses more frequently. It can also be administered by rectum by the Murphy drip method. Finally, it can be given intravenously according to a method which I developed.

The intravenous method seems of importance because the drug is particularly indicated in uremic conditions with vomiting, delirium and disturbances of the sensorium in which theobromin cannot be given by mouth, or if so given, the amount absorbed is very slight or nil. I use a 5 per cent. solution, the percentage ordinarily used in animal experimentation. Pharmacological examinations show that the specific gravity of a 5 per cent. solution at 20°C. is 1.0228, and its alkalinity equivalent to that of a 2.4 per cent. solution of sodium bicarbonate. Sodium bicarbonate in this concentration, or even stronger, is sometimes given intravenously in diabetic coma. A 5 per cent. theobromin sodium salicylate solution heated in a closed vessel at 96°C. for one hour, and then for another half hour shows slight yellowish discoloration and a very slight loss of alkalinity, but remains perfectly clear. Sterilization does not alter the solution. The dose I usually employ is 15 grains, *i.e.*, 20 c.c. of 5 per cent. solution. In one instance, 30 grains (40 c.c. of the solution) were given in a single dose. The stock solution should be resterilized by boiling immediately preceding each injection. If the entire solution enters the vein, there is no local reaction. If a few drops find their way into the subcutaneous tissue, some induration, ecchymosis, or slight pain lasting a few days sometimes follows. In one instance, through a misunderstanding by one of the house staff, the solution was injected subdermally; a local skin slough and ulcer resulted. The injections are never followed by systemic reactions. The solution is readily prepared and sterilized. While 20 c.c. of a 5 per cent. solution is a convenient standard, the dosage can be modified to suit individual requirements. The ordinary dosage can be given intravenously daily for several days.

It is known that the diuretic function of the kidney becomes fatigued by long-continued theobromine administration, hence it is advisable to give the drug in the customary oral method in courses of two or three days with intermissions of similar periods. Theobromine sodium salicylate (diuretin)

is particularly indicated in the dropsy of cardio-renal disease. Given on alternate days with digitalis, it often enhances the action of the latter, not only in cardio-renal but also in purely valvular disease. As an adjuvant, theobromine is often combined with the Karrell diet. Because of its importance, this diet and the method of its administration require detailed description.

The **Karrell Diet** consists in daily administration of 800 to 1000 c.c. of milk; it is preferably given in glassfuls of six ounces every three hours. This is usually termed a "Karrell day." On Karrell days the patient should be in bed or at rest. The milk should be sipped slowly. Patients rarely complain of thirst, but occasionally become so hungry that they suffer from hunger pangs and faintness. Under these conditions, I allow them dry toast, crackers, perhaps also an apple or orange in amounts just sufficient to curb extreme hunger. An excellent routine procedure in moderately severe edema of cardio-renal origin is Karrell diet and theobromine sodium salicylate for two days, alternating with digitalis, and an appropriate renal diet (Chapter XXI) with somewhat restricted fluid intake for the succeeding two days. In most cases, I believe it advisable to continue theobromine therapy and Karrell diet at intervals, long after edema has disappeared and the patient feels well. These intervals should be gradually lengthened; for example, Karrell diet and theobromine may be given for a day, at first twice weekly, then once weekly, then once monthly. In this manner it seems possible to retain and to continue for months, and occasionally for years, the improvement gained by the initial, more intense therapy. In some instances of cardio-renal disease with extreme anasarca, the intensive treatment outlined—digitalis, theobromine and Karrell diet—yields remarkable results: Losses of from 10 to 15 pounds during the first week, and from 20 to 40 pounds in several weeks, are not exceptional. Coincident with reduction of edema, other serious symptoms such as semistupor, dyspnœa, cyanosis and manifestations of hypertension often decrease or disappear. Beneficial results derived from this therapy are most marked in those in whom myocardial insufficiency is a prominent clinical feature.

As a modification of the Karrell diet and as a method of promoting diuresis in patients in whom the urinary output is approximately normal in amount, I have occasionally restricted the fluid intake to but 500 c.c. daily. The liquids allowed are water, sweetened or unsweetened lemonade, and coffee and tea with very little milk. When necessary, only sufficient solids of the kind above mentioned are added to control severe hunger. Patients rarely complain of thirst; when present, it is partially relieved by small quantities of cracked ice, or by rinsing the mouth with water. I give this restricted and modified Karrell diet for two days, with theobromine sodium salicylate in 7 to 15 grain doses to the amount of 45 grains daily. Then follow two days of digitalis with appropiate cardio-renal diet and a slightly restricted fluid intake. By this method, I have occasionally achieved results

which did not follow the ordinary Karrell diet. Older patients with long-standing edema frequently voided from 60 to 80 ounces, and occasionally 100 ounces, on the day succeeding the second theobromine day, the usual time of most marked diuretic action. Coincidentally, edemas quickly cleared up, and cyanosis and especially dyspnœa very rapidly and markedly improved. These secondary results may be due not only to the elimination of water, but also to simultaneous elimination of toxic substances.

**Strychnine.**—In animal experiments, strychnine has been shown to have a vaso-constrictor influence, and hence has been assumed to be of value in low blood pressure and shock. However, careful observations of its effect on the circulation in health and in cardiac disease have shown no influence upon the urinary output, edema, blood pressure or heart rate—the usual criteria of any effect on the circulation. The claim, formerly made, that in some intangible way strychnine acts as a circulatory "tonic" by its influence upon the nervous system is not substantiated by any clinical evidence. It may, however, act as a "nerve tonic" in the sense of promoting a feeling of well-being and of lessening the fatigue that often accompanies cardiac disease.

**Vasodilators.**—These consist mainly of nitroglycerin, nitrate of potassium and sodium, amyl nitrite, erythrol tetranitrate and mannitol hexanitrate. Amyl nitrate is the emergency drug of this class. Nitroglycerin, the drug most often prescribed, acts as a peripheral arteriolar dilator. The usual dose is $\frac{1}{100}$ grain three times daily. It may be given hypodermically or by mouth. In circulatory disease it is chiefly used for lowering blood pressure in hypertension and for the relief of precordial pains. It is believed that the latter is accomplished by the relief of spasm of the coronary arteries. In some cases of hypertension I have administered as much as $\frac{1}{10}$ of a grain of nitroglycerin subcutaneously three times daily with little or no effect upon hypertension or precordial symptoms. These and other inconstant results following nitroglycerin may be accounted for by the various amounts of arterial thickening and consequent variations in dilatability of the arterioles. In edema of the lungs and in hypertension and nephritis, nitroglycerin in large doses (from $\frac{1}{25}$ to $\frac{1}{10}$ gr. hypodermically), combined with the usual cardiac remedies, occasionally relieves the overburdened heart by its effect upon the peripheral circulation.

Nitroglycerin is of most value in cardio-sclerosis with moderate hypertension and labile vasomotor mechanism (Chapters XVII, XXIII). Its administration may then result in fairly long continued lowered blood pressure and in relief of symptoms. If the use of nitroglycerin be limited to such cases, it becomes a very valuable drug. If, however, it is used in continued hypertension with advanced coronary disease, nitroglycerin is apt to be followed by disappointing results for reasons already mentioned. Erythrol tetranitrate and mannitol hexanitrate have a slower and more continued action than the others of the vasodilator group.

*Aromatic spirits of ammonia* in teaspoonful doses, *Hoffman's anodyne*, in one half teaspoonful doses on sugar or in water, and *benzyl benzoate* in one half teaspoonful doses in water are drugs of occasional value in combating some of the troublesome flushes and occasionally also some of the precordial pains found in those with hypertension and vasomotor symptoms.

**Camphor.**—This drug, especially in the form of camphor in oil, given hypodermically, has been a favorite circulatory remedy for many years, especially on the continent. Careful clinical observation, however, has shown that it has no demonstrable influence upon the circulation, for it exerts no influence on diuresis, blood pressure or pulse rate. In animal experimentation there is a slight effect upon the pulse pressure. The use of spirits of camphor in depression of the nervous system, as, for example, in fainting spells, is sometimes of value because of its reflex action emanating from the local effect of camphor on the gastric mucosa.

**Alcohol.**—Discussion of alcohol is here limited to its effect upon the circulation. In animals, alcohol slightly increases cardiac contractility. When given in large quantities in human beings, alcohol produces a marked fall of blood pressure by its depressant action upon the vaso-constrictor center and upon the heart muscle. Small quantities of alcoholic beverages occasionally seem to augment cardiac contractility in decompensated cardiac disease, but the effect is slight and inconstant, and its value problematical.

**Aconite.**—Recent investigations have shown that the strength of the tincture of aconite, the preparation usually used, varies considerably, and that, even when properly standardized, the dose ordinarily administered is much too small. Aconite does not slow the heart rate in cardiac disease. Its presumed therapeutic value is based upon experimental investigation in animals in which cardiac rate, contractility and blood pressure are lowered as the result of a central vagus effect. When given in very large doses, the drug sometimes increases ventricular irritability. This has been shown by the production of extrasystoles by pressure over the pneumogastrics in patients who had been given the drug.

**Sparteine.**—This drug is the alkaloid of the common broom. Formerly it was used as a cardiac tonic because it was presumed to have an action like digitalis. When injected into animals, it slows the heart and slightly raises the blood pressure. Clinically, these effects have not been observed; it must be added that careful investigation of its action is lacking. The dose advised ranges from ½ to 5 grains; probably two grains of sparteine sulphate is a safe dose for an adult.

**Suprarenal Extract—Adrenalin.**—Its vaso-constrictor action, especially when given intravenously, is well known. It may be used in circulatory collapse accompanied by a sharp fall of the systolic blood pressure, or in hypertension with rapidly falling blood pressure, the latter indicating myocardial insufficiency. Injection of the drug in these conditions is sometimes followed by decided, though temporary improvement. I have found the drug of

special value in various types of vasomotor instability with or without tachycardia. Adrenalin has also been advised in so-called constitutional hypotension (Chapter XVIII), a condition in which cardiac failure is absent. The main symptoms in these individuals are those referable to vasomotor disturbances. The hypotension itself rarely requires medication, but the accompanying symptoms may be relieved by adrenalin. The best method of administration is the standard sterile solution in the strength of 1 to 1000; the dose is from 15 to 30 minims given internally, or, preferably, hypodermically or intravenously. The drug may also be given in tablet form in one grain doses.

**Morphine.**—This drug produces slowing of the heart rate by its effect upon the medullary center. Injections in dogs are sometimes followed by complete heart block. In man, morphine is a cardiac "stimulant" only in the sense that, by its effect on the central nervous system, the patients become less restive and irritable. Thus regarded, it is often an excellent aid to other drugs in such acute circulatory conditions as paroxysmal tachycardia, acute onset of auricular fibrillation, and sudden cardiac failure with dyspnœa. It should then be given hypodermically in full therapeutic doses. Morphine is almost invaluable in the pulmonary edema that accompanies attacks of coronary occlusion.

**Bromides.**—These are indicated in tachycardia from any cause, or in those in whom extrasystoles cause subjective sensations. In the former, where morphine may also be indicated, the bromides enhance and prolong the morphine effect. Even though the bromides do not reduce the cardiac rate, they are sometimes effective in relieving the patient of many of his subjective sensations.

**Luminal.**—This is a new synthetic drug. I have used it frequently to control restlessness and sleeplessness in cardiac disease as well as in annoying tachycardia from any cause. The usual dose is one and one half grains, given in tablet or powder form once or twice daily. I have also employed luminal sodium in doses of one and one half grains hypodermically in various cardiac conditions accompanied by dyspnœa and tachycardia in which hypnotics like morphine or codeine did not seem indicated. On the whole the results were gratifying. One may also increase and prolong the quieting effect of a small dose of morphine by combining the latter with sodium luminal: Two separate injections must then be given.

Other sedatives which may be employed to combat restlessness and sleeplessness are chloral hydrate and veronal (barbitol); they may be given alone or in combination with small doses of morphine or codeine, or with the bromides. Contrary to the usual opinion, chloral hydrate and barbital have not a sufficient depressant action on the circulation to contraindicate their use.

**Acetate of Potash and Soda.**—Their diuretic action is usually slight and depends upon the non-metallic salt—the acetate—for its effect. The

dose is from 15 to 30 grains, given in solution every two or three hours. It is occasionally of advantage to alternate these drugs with the more active diuretic, theobromine sodium salicylate.

**Saline Cathartics.**—Brisk catharsis is sometimes of value in starting diuresis. The best saline for this purpose is the sulphate of magnesia.

**Calomel.**—The drug is sometimes of benefit in cardiac dropsy; it is of less value in edema of renal origin. The dose generally recommended is the heroic one of three grains three times daily for two to four days. After an interval of a few days, unless some contraindications exists, it may be repeated in the same dosage. Its action is acribed to direct stimulation of the renal epithelium. I have had no experience with calomel given in this manner.

**Venesection.**—This procedure has a limited sphere in plethoric cases with cyanosis, dyspnœa and threatened or actual pulmonary edema. It possesses a double advantage: From the circulatory standpoint, its chief advantage probably lies in a reduction of venous pressure, for the latter is usually abnormally high in the type of patients just cited. Bleeding also removes toxic products. The amount to be withdrawn must be individualized; the usual amount if 400 to 600 c.c.

**Multiple Skin Puncture.**—This procedure is sometimes of great value when massive general anasarca exists for a long time and does not respond to the usual therapeutic measures. The procedure is as follows: The skin of the leg is cleansed with soap and warm water and then washed with a bichloride of mercury solution. For the punctures a rather large-sized, triangular surgical needle (glover's needle) is used. The needle and the physician's hands should be sterile. The needle should be firmly grasped with the fingers one half inch from the needle point. It should then be quickly plunged into the skin, especially of the leg and dorsum of the foot up to the level held by the fingers. From 50 to 100 punctures in each extremity should be made. The punctures must be made very rapidly and should not take more than about two minutes. The legs should then be swathed in large, clean bath towels. It is surprising to see how much fluid can be drained off in this manner. The patients are relieved not only locally but also from the general circulatory standpoint. The skin punctures often give cardiac medication a better change for therapeutic results. To emphasize a necessary modification of the procedure, two cases deserve mention. In one, massive drainage of fluid from both extremities was followed by semi-stupor lasting twenty-four hours; in the other a similar semi-stupor developed into unconsciousness and death in three days. In both, the loss of the fluid *per se* (oligemia) seemed accountable for the symptoms. I would therefore advise, where anasarca and decompensation are marked, that there be an interval of several days between puncture of the two extremities.

Paracentesis of the thorax and abdomen for hydrothorax and ascites respectively has its appropriate place for the removal of pleural and peritoneal exudates when these are due to cardiac failure and cannot be removed by

circulatory drugs. The removal of such accumulated fluid by paracentesis often gives drugs a much better chance for beneficial results.

**Autogenous and Stock Vaccines, and Sensitized Sera.**—I have employed autogenous vaccines in a number of cases of endocarditis due to the streptococcus viridans (Chapter XV), but have not been able to discern any benefit from their use. There was no effect upon the temperature or toxic symptoms, nor was there any change in the clinical course of the disease. In a few cases that came to necropsy in which this therapy was employed, nothing was found to indicate that the vegetative process on the valves was checked in in the slightest degree.

I have also administered stock vaccines in a small number of cases of endocarditis which seemed clinically of streptococcic origin but in whom the organism was not isolated from the blood. Although I observed no ill effects from the injections, they had not the slightest therapeutic result.

In a number of instances of streptococcus viridans infections, I employed serum derived from horses sensitized to the coccus. The effect of such injections was also entirely negative.

**Blood Transfusions.**—Except for the temporary relief of anemia, I have seen no lasting beneficial effects of transfusions in streptococcus viridans infections either in the fever or the clinical course of the case. My experience with transfusions from donors immunized with the streptococcus viridans is limited; in the few cases I observed, there was no appreciable benefit.

**Colloidal Silver Preparations.**—Interest in the use of silver salts in septic conditions has been revived by the administration of various colloidal silver salts in the treatment of acute rheumatic and bacterial endocarditis. I have observed a few cases in which the silver colloids were injected intravenously. In one, a streptococcus viridans infection, twenty injections were given at two and three day intervals without benefit; the patient died with the symptoms of cerebral embolism. In another case, a child with acute rheumatic endocarditis, a few injections were followed by a rather sharp fall in temperature. In the same ward, I had under observation at the time a male adult of twenty who suffered from an acute rheumatic outbreak, and from acute endocarditis and pericarditis with effusion. The cardiac involvement ran a very stormy course: The temperature reached 105° and remained between 104° and 105° for one week; dyspnœa, chiefly due to the very large amount of pericardial exudate, was extreme. The patient received neither silver injections nor serum therapy of any kind. After one week the temperature fell suddenly, signs of pericardial effusion quickly disappeared, the patient rapidly convalesced with the physical signs of a permanent aortic lesion. Thus are contrasted two cases of an acute endocardial infection both of whom rapidly convalesced, one with colloidal silver injections, the other without any attempt at specific therapy. The inference is clear that there is no proof in the first case that the injections caused a recession of the acute endocarditis. In the enthusiastic reports regarding the use of colloidal

silver in endocarditis, I have found no instance of "cure" which could not be explained upon the basis of an acute endocarditis which had quickly run its febrile course, or upon the basis of a longer duration with the usual remissions. In other words, I do not believe that the intravenous use of silver preparations is warranted in any type of endocardial infection.

**Massage—Passive Motion.**—As a result of careful observation of many cases of cardiac disease, it is my conviction that patients are allowed in bed or at absolute rest too long after compensation has been restored or after an inflammatory process in the heart has run its course (Chapter XXII). Two or three weeks after the quiescence of inflammatory symptoms and after restoration of compensation constitute, I believe, the average time for complete rest. After that period the judicious use of massage, active and passive motion, and exercise aid the circulation by slowly increasing heart work, and produce a more rapid return to normal circulatory conditions. No form of exercise or massage should be pushed far enough to cause dyspnœa, dis ress, rapid heart action or unpleasant subjective symptoms. Massage and passive motion also have some value as added therapeutic aids in long continued cardiac decompensation in bedridden patients. The chief contraindication in such individuals is extreme continued dyspnœa. In such cases, massage should be limited to the extremities and to the muscles of the back; the abdomen and the anterior part of the chest should not be included. When edema and venous stasis are present, it may be of advantage to stroke the limbs in the direction of the venous and lymphatic current, *i.e.*, from the periphery to the trunk. This sometimes decreases the local engorgement.

The kind of massage to be employed in cases of restored compensation must be individualized. Usually light superficial massage alone is tolerated. Deep vigorous massage and kneading of the muscles produces discomfort and pain, and, by exciting the patient, may cause rapid heart action and dyspnœa. The amount of passive motion must be similarly studied in every individual case. Flexion and extension of the smaller joints—fingers, wrists, ankles and toes—should at first be practiced gently once daily; subsequently the larger joints (knees, elbows, hips and shoulders) should be flexed and extended. If these procedures are not followed by pain, cyanosis, tachycardia, exhaustion or dyspnœa, more vigorous and oft-repeated passive motion is indicated. This does not necessarily require the services of a trained attendant; in any case, however, it should at the outset be carried out under the direct supervision of the physician, so that he can carefully observe any possible ill effects following its use.

**Calisthenics—Medical Gymnastics—Resistance Exercises.**—These are occasionally indicated while the patient is still in bed. They require the more active cooperation of the patient and make more demands upon the circulatory reserve. Rapid breathing, dyspnœa, increased and prolonged pulse rapidity, or subjective feelings of faintness and exhaustion are the best clinical guides that gymnastics or exercise have exceeded safe limits and have caised

marked diminution of the cardiac reserve power. It should be emphasized that these treatments are only added therapeutic aids, and cannot replace therapeutic drugs when indicated. The use of dumb-bells and of carefully graduated exercises as measured by the ergostat are also of value. Resistance exercises may also be practiced in suitable cases. These consist in the well-known flexion and extension of the wrist, elbow and shoulder joints of the upper, and of the ankle, knee and hip of the lower extremities. Perhaps the Zander apparatus used in various types of joint and neurological disease (mechano-therapy) may also have a special therapeutic application in some forms of cardiac disease. It should be remarked that all types of calisthenics, gymnastics, etc., should be graded and only slowly increased in duration and strength.

Walking is the best, simplest, and most accessible of all the forms of more active exercise. Some years ago, a system of carefully planned and graduated hill climbing with interspersed resting benches was popular on the continent (Oertel treatment), but arbitrary insistence upon fixed distances to be covered or the hills to be climbed leaves out of consideration the only safe and cardinal guide of circulatory endurance, namely, the patient's cardiac reserve power as measured by his subjective sensations, a constantly varying factor. On the other hand, walking at a moderate pace on the level or up a slight incline for a distance or length of time well within the patient's circulatory power is a much safer and more elastic rule, and approaches more nearly the normal demands and mode of life that the individual will later follow.

**Hydrotherapy.**—The usual hydrotherapeutic procedures consist in tepid baths, sponging with tepid water followed by a quick cool or cold sponge over the entire upper parts of the body, tepid or cool shower baths, needle baths, or sprays of water of varying temperatures applied to various portions of the body. Except for very warm baths, after the patient is properly inured to them, the above measures are applicable to any type of cardiac disease in which compensation has been well established. These measures have their special application, however, in a large group of patients who suffer from marked neuropathic tendencies in addition to their organic disease. These individuals are apt to have vasomotor instability, such as hot or cold extremities, frequent flushed, etc. They may suffer from dyspnœa of purely subjective origin not correlated with exercise or with any discernible cardiac change. They complain of indefinite pains in the chest, neck or arms. They frequently suffer from headache and anorexia. In other words, this type of patient, well compensated, suffers from that train of cardiac symptoms found in the so-called cardiopathic individuals, in whom there is no organic disease (Chapter XVIII). Doubtless the knowledge of the presence of organic trouble plays a role in unduly directing the patient's attention to otherwise trivial symptoms. To some extent, also, I have found that this neuropathic tendency has been caused by consultation with physi-

cians who have exaggerated the importance of the physical ailment and have not sufficiently relieved the mental disquietude and fear of sudden death which exists in many patients with cardiac disease. Naturally, where pathological changes are present, it will require careful discrimination between the so-called neurotic symptoms and those emanating from the organic lesion (Chapter XIV). In those in whom the diagnosis of an added neurotic ailment has been established, the hydrotherapeutic measures already outlined exercise a very favorable influence. Regarding the special procedure to be employed in individual instances, it is necessary to remember that in no case should early treatments be so brusque or vigorous as to frighten or in any way upset the patient. Tepid baths or sponges should be employed at first; later, more vigorous measures—cool sprays and showers—may be used. In this manner the neurotic symptoms, which are very real and disheartening to the patient, may gradually disappear.

*Hot baths* race the heart, hence they should not be used therapeutically in any type of valvular disease in which there is actual heart failure or any tendency to decompensation. There are, however, many stabilized cardiac cases who (carry on) as well as normal individuals. For these, ordinary hot baths have no more than the usual effect in normal people.

**Carbon Dioxide and Oxygen Baths—Nauheim Baths.**—In a different therapeutic category belong those baths in which the water is naturally or artificially charged with carbon dioxide or oxygen. Carbon dioxide is the gas usually used. The gas bubbles surround the body of the patient, produce a sensation of warmth and cause dilatation of the superficial vessels. The claim is made that by the use of these gas impregnated baths, congestion is relieved, the pulse rate slowed, dyspnœa decreased, and the overburdened heart correspondingly benefited. There are conflicting reports regarding the effect of these baths upon blood pressure; it is raised according to some, lowered according to others. In those I have observed, there was no constant rise or fall of the systolic blood pressure following the baths. There seems little room for doubt, that, when taken at spas and health resorts, gas impregnated baths are of benefit. However, this is not so much because of their direct effect upon the circulation, but because of the difference in environment; the enforced rest from business and worry; the more moderate diet, and because of the more regular and physiological mode of living. The baths act upon the circulation in the same manner as any mild stimulating exercise, and, in my opinion, on this alone rests their efficacy on the circulation. Their use should therefore be limited to well-compensated patients or to those with but mild disturbances of compensation. Even here, the baths should not be prescribed universally, but only to those in whom observation has shown a good reaction during and following the bath. Cold extremities, a sense of exhaustion, rapid breathing, accelerated pulse rate, dyspnœa, constitute distinct contraindications. I regard the possible effect upon the blood pressure as of less importance.

The usual routine of a series of carbonated baths given at Nauheim consists of a first immersion lasting from five to seven minutes in a bath of the temperature of 95°F. The temperature of the succeeding baths is gradually reduced to about 86°F.; the immersion time is slowly increased to 20 minutes. The bath is omitted every third or fourth day. A full course consists of two series of twenty baths each, with an interval of three or four weeks. Oxygen and "Nauheim" carbonated brine baths can be prepared at home by dissolving salts, producing oxygen and carbon dioxide gas respectively, in the proper quantity of water. A simple method is as follows: A tub is filled with a sufficient amount of water at a temperature of 98°F., ten pounds of ordinary salt and four pounds of bicarbonate of soda are separately dissolved in a sufficient quantity of hot water. These are added to the bath. The patient then enters the tub. A well stoppered eight ounce bottle containing four ounces of hydrochloric acid is then held by the attendent at the bottom of the tub and as far as possible away from the patient's lower extremities. The bottle is then slowly uncorked under water so that the patient is not burned by the escaping acid. In this manner a Nauheim bath may be safely and readily prepared at home. Naturally the greatest disadvantage of such home baths is that there is no change of environment, with all its attendant and important beneficial features. However, in the group of cardiac patients already described who suffer largely from neurotic in addition to organic complaints, gas impregnated baths, even when taken at home, may be followed by excellent results.

**Types of Patients Suitable for Spa Treatment.**—The question frequently and insistently arises in private practice as to the class of cardiac patients who should be sent to the continent for "Nauheim" treatment. Nauheim is mentioned because it is the most popular and best known among patients themselves. Until some American health resort becomes as popular and can offer the same or similar well-balanced facilities for restful environment and medical supervision, the question must be fairly met and intelligently considered. The answer must depend, not upon the type of valvular lesion or cardio-vascular disease nor upon the degree of hypertension, but chiefly upon the state of cardiac compensation. Patients severely decompensated—for example, those suffering from marked edema, cyanosis and dyspnœa— should be advised against taking a trip for Spa treatment. They are more benefited by proper treatment at home. There are some patients, however, otherwise intelligent, who will go to almost untold risks and expense to achieve what they conceive to be a life saving cure, and this directly against medical advice. I have observed several such patients who returned home worse than when they left; very few were improved. One of those not benefited was a physician with marked cardio-sclerosis, edema and cyanosis, who left for Europe against advice; he returned home a few months later *in extremis*. The patients I have found most improved were those who, besides mild decompensation, were nervous, high strung, or worried, and who at the Spas were

able not only to have enforced rest and quiet, but also to quickly divorce their minds and thoughts from domestic and business cares. It is to individuals of this type that Spa treatment should be recommended with the great probability that it will be followed by good results.

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## CHAPTER XXI

## TOBACCO—DIET—RENAL AND BLOOD TESTS IN CARDIO-VASCULAR DISEASE

**Tobacco.**—The effect of tobacco as a possible causative factor of organic cardio-vascular disease has already been dealt with (Chapter V). It probably produces a fleeting rise of blood pressure. When precordial pains (Chapters XXIII, XXIV) are a feature of the disease for which the patient seeks relief, and smoking has been immoderate, it should be stopped entirely. When precordial pains are infrequent and not severe, and the patient feels the interdiction of smoking a hardship, smoking of an occasional cigar or a few cigarettes daily or an occasional pipe may be permitted. When the organic disease is stabilized and quiescent and is not accompanied by precordial pains, I am in the habit of allowing the patient a moderate amount of tobacco. One should not be unnecessarily arbitrary in chronic diseases but should individualize and weigh comforts from the patient's standpoint against an assumed hastening of the valvular, cardio-sclerotic or arterio-sclerotic process from tobacco, a questionable arterial "poison" at best, especially when tobacco is used in moderation.

From the dietetic standpoint, and because of the frequent correlation of cardiac with renal disease, it is necessary to make a clinical distinction between purely cardiac cases with no or very slight secondary renal involvement, and those in whom renal involvement is the primary or predominant feature. It is also necessary in both types to remember that we are dealing with chronic conditions, and that the dietetic regime will have to be carried out for months and even years. Hence the diet should not be unnecessarily harsh or restricted, and consideration should be given, as far as possible, to personal idiosyncrasies and tastes.

In patients with compensated valvular disease with or without hypertrophy, without renal involvement or hypertension, it is not necessary to make any radical change from the usual dietary of balanced rations of an individual in health. There is no necessity for strict observance of any special diet, nor for the limitation of fluids. On the other hand, there should be sensible restriction about excesses of any kind of food or drink.

**The Use of Coffee, Tea and Alcoholic Beverages, in Cardio-sclerosis.**—To patients who are accustomed to small amounts of alcohol in the form of beer or light wines, I am in the habit of allowing a small amount of these beverages, provided observation shows no deleterious effects upon the pulse rate and blood pressure. I limit the amount to one glass of beer or to a small glass of light wine taken at the principal meal. I have observed such patients for years, and have found no ill effects upon the cardiac condition

ascribable to this practice. Some patients, especially the elderly, find that they fall asleep more readily if they take a small amount of whisky or wine at bedtime. Here also I rarely stop this practice unless I find definite contraindications, either from the cardio-vascular or gastro-enteric systems. Similarly, if patients are in the habit of taking tea or coffee without any ill effects, a cup of weak tea or coffee may be allowed daily. When patients demand much coffee, various types of caffein-free coffee may be substituted.

**Diet in the Obese.**—In obese individuals with cardio-vascular disease, a systematic although gradual attempt should be made to reduce weight. Since these individuals as a rule eat too much, indeed, they often eat gluttonously, it is sometimes sufficient to simplify and lessen their dietary so that it becomes sub-caloric, especially with reference to fats, sweets, pastries and other carbohydrates. If these simple restrictions are not sufficient, saccharine for sweetening purposes, may be used instead of sugar. It should be remembered, however, that some patients have always been fat even though they eat but sparingly. This probably depends upon some endocrine disturbance or idiosyncracy of metabolism. Such individuals should not be reduced much unless there exists some definite indication from the cardio-vascular standpoint (for example, edema, pulmonary or hepatic congestion).

The general problem of reduction is much simpler in purely valvular cases than is those with hypertension or cardio-renal disease. In the latter, there may be metabolic changes which may require serious restriction of the caloric intake of proteins, so that if carbohydrates and fats are also restricted, there may result not only a loss of weight but also a considerable loss of strength. In such individuals it is therefore necessary to study the case from all angles—functional kidney tests, examination of the blood, etc. (q. v.)—before reduction is attempted.

When digestion is good, the following vegetables are suggested as substitutes for carbohydrates and fats: Cabbage, spinach, onions, salad (lettuce, romaine, etc.), beans, carrots, mushrooms.

Those with myocardial insufficiency and dyspnœa without edema are usually too ill to relish a normal amount of solid food, so that the patient's own limitations and dietetic inclinations are usually sufficiently safe guides. But in view of the possibility of visceral stasis and of edema, the fluid intake should be somewhat restricted; about 500 c.c. in addition to solid and semi-solid nourishment is a safe limit. In cases of myocardial insufficiency with edema, pleuritic transudates or marked visceral congestion, a strict Karrell diet for several successive days or in courses of several days each, as already outlined (Chapter XX), is an excellent aid to drug therapeutics. The Karrell diet reduces the salt intake to a minimum. Even after disappearance of edema, and when comparison of the salt intake with its output in the urine demonstrates a normal relationship and no chloride retention, only a moderate amount of salt should be allowed in order to overcome any tendency to edema by salt retention.

In those cardio-renal cases in which the kidneys demand special attention, a brief account of some of the modern methods of kidney study and diagnosis, and their correlation with diet and therapy is required. In addition to the routine examination of the urine for normal and abnormal chemical and microscopical constituents, other methods have come into use to estimate renal function and efficiency; these depend on the power of elimination of certain substances or foods taken internally or given subcutaneously. The status and value of some of these tests and their correlation with clinical data have not yet been definitely fixed. Two of these tests consist in the study of iodide of potash excretion, and of the elimination of lactose injected intravenously. Both of these have been discarded. The first test requires a long time for its completion and gives inaccurate results; the lactose test is too cumbersome and is likewise inaccurate. Probably the most important method at the present time consists in the estimation of the excretion percentage in the urine of a dye which is injected subcutaneously. The dyes formerly employed were methylene blue, indigocarmine and rosanilin. Phenolsulphonephthalein is now almost universally chosen for this study. Briefly, the method is as follows:

Twenty minutes to one half hour before the injection, the patient is given 200 to 400 c.c. of water. The patient is then catheterized, the time noted, and the catheter kept in place. Then one c.c. of a carefully prepared sterile solution, containing 6 m.g. of the dye (phthalein) is injected intramuscularly in the lumbar region. The urine is allowed to drain into a test tube which contains a drop of a 25 per cent. sodium hydroxide solution, and the time of appearance of the first pinkish tinge is noted. The catheter is then withdrawn and the patient is asked to void into two different receptacles at the end of one and two hours respectively. Sufficient 25 per cent. sodium hydroxide solution is added to make the urine decidedly alkaline. This changes the yellowish or orange color of acid urines to a brilliant purplish red. Sufficient water is now added to each specimen to make 1000 c.c. The solution is mixed; a small portion is then filtered and compared with the standard color. In those patients seen in office consultation where the phthalein test seems of value, I have employed the following routine: The patient is asked to void; he is then given a glass of water and the dye is injected. He is sent home, or follows his usual occupation, and is asked to void his urine two hours after the injection; this specimen is then examined for the percentage of dye excretion. I have found this method sufficiently exact for ordinary purposes; it obviates the necessity for catheterization, and requires very little time. In addition, it gives information of dye excretion during the time the kidneys are under normal strain, for the patient is about and not resting in bed. I consider this a very important consideration in ambulatory cases.

As colorimeters the Dunning, Hellige or Duboscq may be used; or one can manufacture a practical and sufficiently accurate colorimeter by

using a series of bottles or tubes which contain the solution of phenolsulphonephthalein in multiples of 5 per cent.; the tubes are sealed and their respective percentage labeled; these may then be used as the color standards. If the dye first appears in the urine within fifteen minutes of the time of its injection, and 40 to 60 per cent. is excreted within two hours, the test is regarded as normal. For clinical purposes, the two hour excretion test alone is sufficient; this saves catheterization. In renal congestion secondary to circulatory disturbances and in contracted kidney with hypertension the percentage and output in two hours is usually decreased. It requires clinical examination to determine which of the two factors—renal congestion or primary nephritis—is the cause of the decreased phthalein output.

For the purpose of testing quantitatively for urea, salt and water output in the urine, a balanced diet containing a measured amount of solids (starches, sugar, fats, proteids and salts) and of fluids (milk, water, tea) is given. Schlayer's diet or some modification may be employed. An excellent and well-balanced ration is that of Mosenthal for testing the renal function. It is as follows:

NEPHRITIC TEST DIET

All food is to be salt free.
Salt for each meal is furnished in weighed amounts.*
All food or fluid not taken must be weighed or measured after meals, and charted.
Allow no food or fluid of any kind except at meals.
Note any mishaps or irregularities that occur in giving the diet or collecting the specimens.

BREAKFAST, 8 A.M.

Boiled oatmeal, 100 gm.
Sugar, ½ teaspoonful.
Milk, 30 c.c.
Two slices of bread (30 gm. each).
Butter, 20 gm.
Coffee, 160 c.c.
Sugar, 1 teaspoonful } 200 c.c.
Milk, 40 c.c.
Milk, 200 c.c.
Water, 200 c.c.

DINNER, 12 NOON

Meat soup, 180 c.c.
Beefsteak, 100 gm.
Potato (baked, mashed or boiled), 130 gm.
Two slices of bread (30 gm. each).
Butter, 20 gm.
Green vegetables, as desired.
Tea, 180 c.c.
Sugar, 1 teaspoonful } 200 c.c.
Milk, 20 c.c.
Water, 250 c.c.
Pudding (tapioca or rice), 110 gm.

SUPPER, 5 P.M.

Two eggs, cooked in any style.
Two slices bread (30 gm. each).
Butter, 20 gm.
Tea, 180 c.c.
Sugar, 1 teaspoonful } 200 c.c.
Milk, 20 c.c.
Fruit (stewed or fresh), 1 portion.
Water, 300 c.c.

*One capsule of salt, containing 2.3 gm. of sodium chloride, is furnished for each meal. The salt which is not consumed is returned to the laboratory, where it is weighed, and the actual amount of salt taken is calculated.

8 A.M.—No food or fluid is to be given during the night or until 8 o'clock the next morning (after voiding), then the regular diet is resumed.

Patient is to empty bladder at 8 A.M. and at the end of each period, as indicated below. The specimens are to be collected for the following periods in properly labelled bottles:

8 A.M.—10 A.M.; 10 A.M.—12 N.; 12 N.—2 P.M.; 2 P.M.;—4 P.M.; 4 P.M.— 6 P.M.; 6 P.M.— 8 P.M.; 8 P.M.— 8 A.M.

The amounts, specific gravity, and sodium chloride content, and the urea determination by the hypobromite method, or preferably the nitrogen content by the Kjeldahl method are separately determined for each two hour specimen, and also for the night specimen. In other words, there are six determinations for the day, and one for the night. In this manner, besides other data, a comparison of the amounts and specific gravity of the day and night urine is made. If the specific gravity is low and shows only slight variations in the different specimens, and the night urine is larger in amount than that voided during the day, it speaks for the presence of a chronic interstitial nephritis. A less exact, yet fairly accurate method consists in measuring together the amounts and specific gravity of the 12-hour day and the 12-hour night specimens. Conclusions similar to the above may be drawn even from this simple procedure.

Outside of hospitals, as in sanatoria or at home, a similar dietetic regime can be carried out if, instead of quantities measured in grammes and cubic centimeters, their known equivalents in cupfuls, glasses and teaspoonfuls can be substituted. This is a modification which I have carried out where careful weighing of the various food constituents could not be done, and I have found the results sufficiently accurate for clinical purposes.

Because of evaporation by the lungs and elimination of water by the skin and intestines, the amount of urine is normally less than the fluid intake. Approximately all sodium chloride, and about 90 per cent. of the nitrogen ingested is excreted by the urine. Thus, the chemical urine tests described give valuable information regarding the retention of salt and nitrogenous products in the body.

In order to determine which of these functional tests is of most clinical value, I made a correlated study of the non-protein nitrogen in the blood, the estimation of carbon dioxide tension of the expired air by means of the Fredericia apparatus, and the phthalein output in a small series of cases. I also observed the alkali tolerance of the urine by giving the patient measured amounts of bicarbonate of soda until the urine became alkaline. Although I have not arrived at any definite conclusions, I believe that a very careful study of the clinical phenomena, of the amount and concentration of the night urine, and of the ordinary examination of several specimens of urine taken within 24 hours, will often allow us to judge and foretell with fair accuracy, what the chemical blood examination (see below) will show with reference to the amount and type of the retained excrementitious products, namely,

non-protein nitrogen and uric acid. In other words, clinicians may soon hope to reap the advantages of the careful scientific work of the laboratory by comparison of its results with ordinary bedside methods.

Chemical examination of the blood has assumed great importance, especially since simpler and accurate methods have replaced the former cumbersome and less accurate determinations. The important constituents sought for are non-protein nitrogen, creatinin and uric acid. The knowledge thus gained is not yet fixed or positive, but there seems to be a correlation between an abnormal amount of non-protein nitrogen retained in the blood, a decreased phthalein output, and clinical evidence of a severe nephritis of a predominantly vascular type. Of special importance are the facts recently brought out that an increase of uric acid in the blood seems to offer an early diagnostic sign of incipient nephritis; that a blood creatinin of over 5 milligrams per 100 c.c. is fatal within a comparatively short time, and that blood acidosis as determined by the Van Slyke method is a valuable index of nephritis both from the diagnostic and therapeutic view points (Chase and Meyers). Not only has our diagnostic and prognostic knowledge been enhanced by these blood examinations, but the dietary of nephritis has assumed a less rigorous and more rational and scientific aspect. The subject of salt restriction has already been mentioned. In nephritics with abnormally increased amounts of non-protein nitrogen in the blood, a low protein and high carbohydrate diet is indicated. The amount of fluid should be restricted if urine measurements show decreased elimination of water. If the amount of urinary excretion is normal, it is advisable occasionally to allow large quantities of fluids—as much as several liters within 24 hours—in the attempt to flush the system and thus get rid of toxic products.

In nephritis with excessive and abnormal amounts of non-protein nitrogen in the blood, attempts have been made to therapeutically attack the disease upon a chemical basis. Sugar solutions have been administered intravenously or by the Murphy drip. In cases of lessened blood alkalinity (so-called acidosis), I have occasionally given intravenous injections of 500 c.c. of a 5 per cent. solution of bicarbonate of soda. One instance was that of a patient of 64 with cardio-nephritis, regular pulse, hypertension, general anasarca and nocturnal dyspnœa, who had been ill over one year. Therapy, consisting of digitalis, theobromin, appropriate diet and occasional Karrell days over a period of several weeks had only a very slight influence upon the symptoms and course of the disease. 500 c.c. of blood were withdrawn by venesection and the same amount of a 5 per cent. bicarbonate of soda solution injected. Within twenty-four hours improvement began and continued uninterruptedly. At the end of two weeks, edema and dyspnœa had entirely disappeared. In another case, a man with cardio-vascular disease, a systolic blood pressure of 250, aortitis, very marked left ventricular hypertrophy, and attacks of nocturnal dyspnœa relieved only by morphine, a similar procedure was employed. The injection was followed by improvement for several

days. In less urgent cases I have tried venesection combined with a 10 per cent. bicarbonate solution per rectum given by the Murphy drip. In all therapeutic attempts of this type to combat acidosis it must be remembered that relief may be only symptomatic and temporary because the underlying kidney condition is often uninfluenced and because abnormal chemical constituents may be present not only in the blood but they may be locked in the tissues themselves.

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## CHAPTER XXII

## SOCIOLOGICAL ASPECTS OF HEART DISEASE—FOCAL INFECTION—PROPHYLAXIS—GENERAL MANAGEMENT—OCCUPATION FOR "CARDIACS"—CARDIAC CLINICS

Including cardio-vascular disease in the term heart disease, it is acknowledged that heart disease is widespread and is responsible for a large proportion of deaths in the community (Fig. 275). Statistics vary but it is evident that valvular disease is prevalent even at the school age, and that cardio-vascular disease is especially prevalent from the 40th to the 60th years. The etiological factors may be grouped generically under infections, intoxications and improper mode of living. This phase alone of the subject presents many aspects and angles. A few of the etiological factors may perhaps be intelligently combatted; others are unknown or are recognized only after the damage has been done to the circulatory apparatus; again, others have given rise to the most diverse, diametrically opposed opinions. I shall briefly sketch some of the practical difficulties of the problem, indicating some controllable, some debatable, and some of the unknown causative agencies. We know, for example, that both lead poisoning and syphilis (Chapter V) especially the latter, can cause cardio-vascular disease. Progress has already been made through educational campaigns in schools, factories, shops, amid lead workers, etc., which have probably tended to decrease the ravages of these factors. This is especially true of lead; but much more educational work will certainly be necessary before syphilis can be classed as a definitely controllable factor. Improper food, especially an improperly balanced, high protein diet, may have a certain influence in causing arterio-sclerosis; but in spite of some experimental evidence, further intensive, exhaustive and long-continued clinical study will be required before we have positive evidence that improper feeding alone is one of the fundamental causes of cardio-vascular disease. Alcoholism can undoubtedly cause cardiac disease, especially myocarditis. As already pointed out (Chapter V), its role in this respect has been exaggerated. There are hopes that alcoholism, even if not entirely eradicated, will gradually become an increasingly minor cause of heart disease. Diphtheria was undoubtedly a frequent cause of cardiac disease and death. Fortunately, toxic cases are now infrequent, and the immediate use of diphtheria antitoxin cures even some of these. Hence diphtheria has dwindled to a small etiological factor of heart disease. Scarlet fever, whether directly or because of the accompanying tonsillitis, may be taken as the type of a frequent infectious cause of valvular disease. We possess no specific remedy that can lessen the probability of the poison

affecting the heart; our efforts are practically limited to the usual routine hygienic and symptomatic procedures which need not be discussed here. Yet, after all, the danger to the heart in scarlet fever is but a small phase of the larger problem of communicable diseases with which preventive and epidemiological medicine must cope. Overcrowding, sanitation, education of the native-born and foreign population represent some of the sociological aspects of the same problem.

**Rheumatism** (Chapter XV)—especially the acute, articular variety—and chorea and tonsillitis are undoubtedly the largest and most important causes of cardiac disease in the middle-aged and young. Here, likewise, we possess no specific remedy which, so far as known, will minimize the danger of endocardial involvement. It is not necessary to enter again into the extremely debatable question of the bacterial origin of the disease. Even assuming this etiology, we have no anti-bacterial remedy. We possess many drugs for the control of the rheumatic articular manifestations, but for the present we can only assume that these drugs thereby lessen the liability to rheumatic endocarditis. Attention of the public should be called to the dangers of rheumatism, they should become acquainted with such simple measures as we possess to prevent rheumatism; these consist chiefly in proper clothing, and protection against rain and snow in the changeable climate of the temperate zone. It may even be necessary to suggest climatic changes in order to obviate rheumatic recurrences. It is not alone the marked cases of rheumatism but also the so-called "growing pains" of children which, in educational campaigns, should be emphasized as evidences of actual rheumatism. The connection between rheumatism, and the tonsil and teeth will be discussed later.

**Tonsillar Infections and Tonsillectomy.**—The inflamed tonsil is the next most frequent etiological factor of endocarditis. Only a short half dozen years ago, the tonsils were eradicated upon the slightest evidence of rheumatism or suspicion of endocarditis. The views at present are not quite as radical. Tonsillectomy has assumed much importance since the demonstration that the tonsils are often the portals of entry for endocardial infections. It is extremely difficult to follow dogmatic rules regarding tonsillectomy in the attempt to prevent the recurrence or occurrence of endocarditis. I have found it necessary to individualize, although I do follow certain general principles. Thus tonsils which are enlarged, ragged and interfere with breathing should be removed: These represent grossly diseased tonsils. If a patient with heart disease be harrassed with frequent tonsillitis, tonsillectomy should be advised, no matter what the size or appearence of the tonsil, or whether the submaxillary glands at the angle of the jaw be enlarged or not. I mention these glands because some assume that these are a sign of chronic tonsillar infection. Maxillary glands become infected from so many other causes, particularly from nasal catarrh, that I do not consider them a valid index of tonsillar disease. If a patient has small,

normal looking tonsils, and has only infrequent attacks of tonsillitis, and the valvular disease has reached a quiescent stage, I advise against tonsillectomy. I do not believe that removal of the tonsils prevents valvular reinfection in such individuals. This view is not invalidated by the fact that tonsils which look normal in the throat show, upon removal, various pathological changes, both macro- and microscopically. Because of their function as filtering agents and because of their constant contact with, and exposure to, bacteria, the tonsils can scarcely ever represent normal lymphoid structures. On the whole, it is still a moot question whether recrudescences of endocarditis have been prevented by the routine and radical practice of tonsillectomy in all children with valvular lesions. Thus, in one excellent report, such tonsillectomies did not affect the frequency of recurrences, nor the course of the disease.

It is important to emphasize that, if possible, the time chosen for tonsillectomy should be during the quiescent stage (Chapter XV) of the cardiac disease. I have seen distinct harm, indeed, recrudescence of the disease, caused by untimely tonsillectomy (see Cardio-vascular Clinics). There are cases, however, in which sore throats—either frank tonsillitis, nasopharyngeal catarrh and so-called rheumatic sore throats—follow each other so quickly that tonsillectomy cannot wait for a quiescence of the endocarditis. In such individuals tonsillectomy, especially in children, is sometimes followed by exceptionally favorable results not only upon the endocarditis but also upon the general condition of the patient. Murmurs may become less loud, their area of transmission decreases, tachycardia and dyspnœa disappear, and what is most remarkable the patients gain rapidly in weight. I have, for example, seen children gain from 10 to 15 or even 20 pounds, so that their appearance changes from frail, anemic children to healthy, robust youngsters. Tonsillectomy here seems to check the infection radically and at once.

Thorough enucleation is the ideal method for removal of the tonsils, so that one can be sure that no tonsillar tissue remains to cause possible reinfection.

**Tooth Infections.**—The teeth have been found to harbor and act as foci of infection in the production of some types of muscular and joint rheumatism, especially in the adult. There have been vigorous campaigns launched both among the laity and physicians to eradicate these foci of infection, and thus prevent rheumatism and heart disease, or, when already present, to prevent their further spread. X-ray examinations of the teeth have been brought as witnesses to show how widespread dental infection may be. It perhaps may be parenthetically, yet pertinently remarked, that dentists can only indicate which teeth are diseased and the type of disease. It remains for the physician to determine the correlation, if any, between the dental infection and the disease in question, and to decide upon the advisability and time of removal of the dental infection. As a matter of personal observation, I believe that diseased teeth as a primary cause of endocarditis is

extremely rare. In the very few I have seen, there was dental caries and septic osteomyelitis of the jaw with general septicemia, endocarditis and death. As the result of careful and long-continued observation, I believe that endocarditis in itself does not warrant more radical or more careful dentistry than is usually required for diseased teeth and purulent foci in otherwise normal individuals. Dentistry here, as elsewhere, should follow sane lines. As with tonsillectomy, I regard routine extraction for the prevention of endocarditis as uncalled for. Extensive extractions upon the supposition that exceedingly small pus foci frequently produce endocarditis is in my opinion unwarranted by general clinical experience and by the negative results following such practice in patients with endocarditis.

The role of pyorrhea has already been discussed (Chapter V).

**Focal Infections.**—The general subject of removal of foci of infection as a prophylactic of, or a curative measure in, heart disease has aroused the medical mind. It is undoubtedly true that a "focus of infection" anywhere in the body, *e.g.*, the inflamed gall bladder, is a potential cause of endocarditis and hence is an etiological factor to be reckoned with. I believe, however, that its frequency as a factor has been tremendously overstated. It is one thing to diagnose a focus of infection; it is another to state dogmatically that its removal is going to benefit the patient. As a matter of fact, it can scarcely affect a cure, for, when the diagnosis of circulatory disease is made, the pathological damage has been done. At best, therefore, eradication of infected foci can either bring only relief or quiescence of the disease. In some older individuals with cardio-renal disease and infected teeth, I saw no effect of any kind from the wholesale extraction of teeth. In other words, prophylaxis against cardio-vascular disease or the presence of the latter rarely calls for more radical surgical intervention and eradication of infected foci than that focus ordinarily demands. To do otherwise is to lose one's clinical balance and to practice medicine by simple rule of thumb rather than by mellowed clinical judgment.

**Occupation and Exercise for the Cardiac.**—Aside from diet and therapy, other questions regarding the management of compensated and decompensated cases of cardio-vascular disease arise. Some of the commoner of these are: Shall a patient return to work? What type of work should he follow? Shall medication be continued and, if so, how long?

With respect to these questions, no matter what the type of lesion there are two preliminary fundamental considerations to be determined, namely: (1) The degree of compensation, and (2) the activity or state of quiescence of the disease. With quiescent compensated lesions, valvular or myocardial in nature, the main restriction regarding exercise should be the kind, rather than the amount, provided always it be well within the patients's cardiac reserve power. This statement requires some modification, however, for the type of cardiac disease plays a role which requires some individual discrimination. For example, patients with tremendous hypertrophic left ventricles

from aortic valvular lesions are scarcely able to maintain long-continued effort without soon encroaching upon their cardiac reserve. In general, it may be stated that even quiescent compensated cases should avoid all exercises which call for competition or for sudden or sharp exertion, as swimming a long distance, running and tennis playing. On the other hand, golf is an excellent form of moderate exercise. It entails the necessity of being away from business and of being out in the open for a number of hours, considerations which in themselves are very desirable. All things considered, walking is the best and simplest form of exercise. In exercise as well as in work it should be emphasized that patients should keep well within their individual limits of fatigue.

The question of occupation and vocation for patients with cardiac disease has recently received wide consideration from the lay as well as from the medical standpoints. It is now generally recognized that many individuals with cardio-vascular disease are not thereby necessarily precluded from attempting to earn a livelihood, and that, if proper work be chosen, they may become self-sustaining members of the community. Occupations and vocations at which patients sit or stand are preferable to those which require walking or stair climbing. Positions in counting houses, clerical work, draftsmanship, light manufacturing industries, working at lathes or small machinery, watchmen, are examples of the work which these patients may safely follow; but just because of these sedentary and easy occupations, exercise out in the open, chiefly walking, should be advised. It is, I believe, a therapeutic error to attempt to avoid all circulatory strain by having these patients pass an almost muscularly inert existence, for it is only by mildly stimulating the circulation by appropriate gentle exercise that the heart and circulatory apparatus are kept at their proper individual level of efficiency. In this respect the heart does not differ from other weakened muscles whose strength is enhanced by moderate, well-planned and individualized exercises. Mild, appropriate dumb-bell exercises and other calisthenics, and breathing exercises should be advised when walking is not feasible, or as additions to the latter.

It has been found that when those with cardiac disease (so-called "cardiacs") find employment, the fact that they are handicapped and hence cannot cope with other workmen in efficiency and earning power does not depress them; on the contrary, they find happiness and satisfaction in being able to earn something which will help them toward becoming independent and self supporting. It is, on the whole, difficult for cardiacs themselves to find the proper type of employment. Hence, in the larger cities, bureaus are established for the poorer and needier, through which appropriate employment may be found. Under such circumstances, the bureau head seeks information from the medical chief of the cardiac clinic and from the social service department (q. v.) in order to determine the functional capacity, the state and type of cardiac lesions, and the home surroundings of the individual. In

this manner, some individuals have been given suitable employment and have been made earning members of the community. In an excellent report on employment for the cardiac by I. M. Duggan, the following was given as a partial list of employments in which cardiacs had been placed:

- Fountain pen factories (male)
  - Grinding pens
  - Polishing barrels
  - Assembling
- Jewelry (wholesale)
  - Chasing (men up to 30)
  - Filing
  - Soldering
- Carding (women up to 50)
- Matching and stringing pearls
- Architect's office
- Draughtsman
- Filing blue prints
- Figuring plans
- Western Electric (only boys, mild cases)
  - Taught business from beginning in each department
  - Draughtsmen and mechanical engineers
- Hotels
  - Door men
  - Watchmen
  - Elevator operators
- Kitchen and pantry
- Linen seamstress
- Assistant housekeeper
- Clerical
- Hand sewing
  - Repairing expensive laces
  - Finishing
- Publishing companies (male and female)
  - Plain clerical
  - Folding and inserting
  - Information clerk
  - Book binding (certain kinds)
- Wholesale grocers (female up to 25)
  - Dictaphone operators
  - Comptometer operators
  - Plain clerical
- Tobacco companies (male not more than 45)
  - Stripping
  - Packing
- Assistant engineer
  - Auditor
- Telephone operator
- Yard man
- Timekeepers
- Vegetable man
- Painters
- Carpenters
- Moving pictures (male under 40)
  - Inspector of reels
  - Clerical
  - Checking
  - Detective and watchman (male up to 60)
- Government property
- Steamship piers
- Private homes
- Hotels
- Construction companies
- Steamship companies
- Checkers on piers (must join union for this work)
- Lamp shades
  - Hand sewing
  - Frame making
- Wholesale meat houses
  - Checkers
- Biscuit Co. (young girls)
  - Light packing
  - Tying cakes
- Piano factories (men up to 60)
  - Assembling small parts
  - Polishers
  - Felt hammer department and gluing
  - Tuners
- Can factories
  - Tinsmith helpers
  - Soldering
  - In lithograph department
- Automobiles
  - Salesmen, supplies
- Mechanic and construction department

Placement for cardiacs has also this important medical aspect, that in some instances it may save patients from attacks of decompensation which are due to cardiac overstrain from improper and too laborious employment. To

that degree, it will decrease hospital admissions from this cause, and save hospital beds and money for other patients.

**Social Service Work and Cardiac Clinics.**—Cardiac clinics are now established in some of the larger cities, not only for group treatment of cardiac disease but also to bring home to physicians and laity the importance and size of the problem involved. The cardiac clinic must not only be thoroughly organized from the medical standpoint, but other activities are necessary and must be organized in order to carry out important sociological work. These will be described first, the clinic itself, last.

There is the social service department, an all important adjuvant to the clinic. Besides information regarding the financial status of the patient and his family, there is correlated socio-medical work as part of its domain. If, for example, the social service head be a trained nurse, she can readily control the group exercises of the cardiac, especially of the children; her medical knowledge is sufficient for her to observe beginning dyspnœa, rapid or overforceful heart action, etc. in those taking part in graded exercises and games. Follow-up work by means of which home conditions are studied, parents and relatives given proper information regarding ventilation, sanitation, clothing, food and exercise (especially stair climbing) is also part of the work of this department. It is the most important link between the clinic and the home; without it, the clinician has no way of determining whether treatment and advice given by him can or will be carried out. If one is dealing with a child, the question of schooling and, with it, the advisability of special school classes must be considered; if with an adult, the problem of proper employment must be carefully discussed. These are but two instances of the vast number of questions upon which the work of the social department touches. When carried out in a kindly, unobtrusive way, this department tends to harmoniously bind the medical with the equally important social problems of the cardiac. In large cardiac clinics, more active educational work may be undertaken through talks given by medical and social attendants to mothers of cardiac children. These should, of course, be simple and should touch upon such topics as sanitation, ventilation, proper diet, rest, exercise, the hopeful aspect of many of the cases, the importance of operative treatment of the tonsils and teeth in appropriate cases, etc.

In well equipped hospitals under whose auspices cardiac clinics are managed, there should exist helpful cooperation. Where possible, frequent consultations between cardiac and correlated departments should be the rule, in order to gain proper viewpoints and well balanced judgment regarding the necessity and type of operative treatment to be carried out. Unless unavoidable because of crowded conditions, mere routine sending of patients to nose, throat and dental clinics is inadvisable.

Where the proper hospital facilities are at hand, X-ray plates or fluoroscopic examination of the chest should be made in order to have data regarding the size as well as the activity of the heart. Electrocardio-

grams are also of value, especially in fixing types of arrhythmias and in aiding the diagnosis of congenital lesions and of myocarditis (Chapter IX).

In order to obviate the necessity of the cardiac child going home for lunch or climbing stairs at school, or even visiting the cardiac clinic, special cardiac classes are established in schools; lunches are supplied, rest periods regulated, and graded exercises and games carried out. When such special classes are not available, cardiac classes can be organized in health centers or settlement houses; under such circumstances a special teacher for educational purposes and a proper nurse are required. The physician can visit and examine the children in their class rooms and give such directions and instructions as he finds necessary.

All these correlated activities are naturally predicated upon the proper formation of the cardiac clinic itself. A concerted attempt among cardiac clinics has been made to standardize and simplify the classification of cardiac disease. The following has finally been adopted:

Class I. Organic (able to carry on habitual physical activity).
Class II. Organic (able to carry on diminished physical activity).
*A*. Slightly diminished.
*B*. Greatly diminished.
Class III. Organic (unequal to any physical activity).
Class IV. "Possible" heart disease (doubtful murmurs: mainly accidental, possibly organic).
Class V. Potential (predisposing history).

One may perhaps cavil at the advisability of some of these groupings. For example, "potential" cardiacs—chiefly children that have had rheumatism, chorea and frequent tonsillar attacks—should undoubtedly be carefully examined from the cardiac point of view, but I fear that overlapping of function can scarcely be avoided if "potential cardiacs" are grouped in the cardiac clinics. A simpler grouping of organic heart disease might perhaps be that of the quiescent, the mild, and the severe insufficiency cases. But for the sake of uniformity among cardiac clinics, the first mentioned classification should be followed as adopted. Once graded, it becomes a comparatively easy task to carry out appropriate treatment. Thus Class III, the severely decompensated, are not ambulatory cases. They belong at home or in the hospital. When again beginning to compensate, the questions of active and passive motion (Chapter XX), and the time for being allowed out of bed must be decided. The first class also—those with physical signs but no symptoms—are readily disposed of. Children and adults may follow the usual conservative daily routine of normal individuals. They may practice mild sports and games; but they should eschew violent sports such as football and should not participate in competitive games. When possible, laborers should choose a type of work that does not require constant extreme muscular exertion. One can only generalize by stating that all

patients with organic cardio-vascular disease should exercise, work or play well within their individual cardiac tolerance. I believe this warning justified in spite of the well known fact that cardiacs are found among laborers and athletes who have never shown any circulatory symptoms. In general, those with markedly hypertrophied hearts (usually aortic lesions) should be more conservatively advised regarding the type of sports and games and regarding such routine as stair and hill climbing. Next in the scale of conservatism are cases of mitral stenosis. One may be least conservative in those of this group (first class) who have quiescent mitral regurgitant lesions.

Class II, the mild insufficiency group, is that which usually constitutes the majority of the ambulatory patients. They likewise offer the best opportunity for improvement by means of graded games and exercises. I refer here to children alone: Adults of this group, if sufficiently improved, should seek suitable employment (p. 401).

Exercise among children is best achieved by graded games and drills. Games that in addition give proper posture are of special advantage. Exercises should be supervised by a doctor, trained nurse or specially instructed social worker. Various types of drills—all rhythmic—may be devised. The simplest are those in which, at the beginning, all may take part. In general, they correspond to mild setting-up exercises, leaving out sharp flexing of the abdomen. Upward and lateral movements of the arms, clapping hands above the head and in front of the body, lateral and forward, lateral and slight backward bending of the body on the trunk, bending of the knees and hips are some of the usual types of drill exercises. Military tramping and shuffling steps, marking time and other exercises will readily suggest themselves.

More active games may be devised for those who have shown no circulatory strain as a result of the milder exercises. Breathlessness, prolonged tachycardia, overforceful and violent heart action, drawn and tired features are some of the more obvious, readily discernible evidences of such overstrain. Such games as hopping, pitching ball, rhythmic dancing, etc. are suggested as more active games in the better compensated cases.

An excellent adjuvant to cardiac clinics is some form of convalescent country home. A sojourn in the country is especially advisable for those who have recently recovered from endocarditic reinfections, or who look pale, tired and undernourished. In the latter, overfeeding should be attempted. To bring these patients to normal weight is perhaps one means of strengthening the entire organism against the inroads of infection. The home should not be too far from the city so that the patients can be occasionally visited by relatives, thus forestalling nostalgia. Convalescent homes are especially appropriate for those with fair exercise tolerance.

I have used the following scheme in my cardiac clinic.

Date 19 Dispensary No. Hospital No. Primary diagnosis Examined by

CARDIAC CLINIC

SOCIAL HISTORY

Name..... Sex..... Age..... Address..... Race.....

| | | Living | Health | Alcoholic | Working | Occupation | Wages | |
|---|---|---|---|---|---|---|---|---|
| For Child | Father | ..... | ..... | ..... | ..... | ..... | ..... | Family income $ Intelligent? Responsive? Clean? |
| | Mother | ..... | ..... | ..... | ..... | ..... | ..... | |

| | | Married | Occupation | Working | Alcoholic | Wages | |
|---|---|---|---|---|---|---|---|
| For Adult | Male | ..... | ..... | ..... | ..... | ..... | Wife living? |
| | Female | ..... | ..... | ..... | ..... | ..... | Husband living? |

Children: Number.....Ages.....Number working.....

Home: T.* P.H.* Number rooms..Number dark.....Rent $....Boarders.....

Sanitary conditions: Neat Untidy Dirty Separate beds.....Patient's room.....

Food: Sufficient? Yes No .....Cooking: Good.....Fair.....Bad.....

Needs.....

Religion.....Church or settlement affiliation.....Immediate needs.....

Sick benefits.....Aid from societies.....Insurance.....

LATER SOCIAL SERVICE REPORT *Date*.....19..

Home: Improved? Yes No Food: Sufficient? Yes No Cooking: Good Fair Bad

Patients: T. P. R. Symptoms.....

Treatment and advice.....

.....

.....

FIRST EXAMINATION—CLINICAL NOTES AND TREATMENT

Family History: Rheumatism.....Chorea.....Cardiac disease....Alcohol .....

Previous History: Rheumatic fever.....Tonsilitis.....Chorea.....

Myalgia.....Purpura or Erythema.....Scarlet fever.....

Diphtheria.....Venereal.....Alcohol.....Tobacco.....

Sleep.....Appetite.....Bowels.....Occupations.....

Other diseases and complications.....

Patient feels.....Patient's statement.....

Appetite.....Digestion.....B'kfast.....Dinner.....Supper.....

Bowels: Move at.....Between meals .....

Sleep: In bed at.....Up at.....Windows open? Rests? to.....to.....to.....

Outdoors.....to.....School hours.....A.M. to.....P.M. Open air class.....

Exercise: Walking Running Playing Stairs: Flights a day.....

Flights at school..... Rest bet. flights.....Lives flights up.....

Dyspnœa? Cough? Pain .....

Palpitation: Subjective? Tachycardia? Edema: Where? When? .....

Color: Good.....Pale.....Cyanosed.....

Nutrition—Weight: Stripped.....Clothed.....T. P. R.

Aspect: Calm—Healthy...Quiet....Active.....Dyspnœic.....Sick....Prostrated.....

Tonsils: Size.....Smooth.....Ragged.....Buried....Crypts: Large....Small.....

Removed: Completely? Incompletely?

Lymph nodes: Cervical.....Size.....Many.....Few.....

Teeth.....Good.....Bad.....Extraction necessary? Pyorrhœa?

* T. = Tenement. P.H. = Private House.

CARDIAC EXAMINATION

Apical impulse: Circumscribed?.....Diffuse?..... { Forcible / Weak / Heaving }

Location of apex: Maximum in.....Interspace.....c. m. from M. S. L.....

Borders: R. c. m. from M. S. L.....L. c. m. from M. S. L.....

Rhythm: Regular.....Auricular fibrillation? Irregular..... Sinus arrhythmia? Tachycardia? Extrasystole? Heart block?

*Sounds:* At apex I.....II.....

At Rt. base, A. I.....II.....

At Left base, P. I.....II.....

Murmurs: Maximal site.....Transmission.....Time of occurrence.....

Quality: Soft? Harsh? Blowing? Rumbling? Musical? Rough?

Effect of inspiration.....Expiration.....

Pulse rate: Standing.....Lying.....After exercise.....

Character of pulse.....

Blood pressure: Brachial { Systolic / Diastolic.....Leg { Systolic / Diastolic.....

Attacks of decompensation: Duration.....Date.....

Chief symptoms.....

Exercise tolerance.....

Present attack: Time of onset.....Initial symptoms.....

Dyspnœa.....Edema.....Pain.....Cough.....

Disability.....Palpitation { Tachycardia? / Subjective?.....Gastric symptoms.....

Loss or gain of weight.....Color.....Urine.....

Pericarditis: Dry.....Friction sounds.....Area.....Time of occurrence.....

Effusion.....X-Ray.....

Lungs.....

Abdomen.....

Liver.....

Spleen.....

Type of lesion: Quiescent?.....Progressive?.....

Electrocardiogram.....

Prognosis.....

.....

Advice.....

Group classification.—See 403.

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## CHAPTER XXIII

## PRECORDIAL PAINS OF CARDIO-VASCULAR ORIGIN—ANGINA PECTORIS—CORONARY DISEASE—ETIOLOGY, PATHOLOGY, CLINICAL COMPLEXES AND THERAPY

Pains due to pericarditis are not included in this description.

Pain in the chest, especially in the substernal region and in the left breast, is such a frequent accompaniment of all types of cardiac disease that, although but a symptom, it requires minute consideration. Precordial pains in all types of severe *organic* cardiac disease is probably due fundamentally to some sudden disturbance in nutrition (as in coronary occlusion), to some added infection (as in rheumatism), or to some adverse mechanical factor (as in cardiac dilatation). These three factors may be summed up in the one that the basic cause of heart pain lies in some marked alteration of the myocardium by which it cannot contract and pump blood efficiently. Even here, the general qualifications are demanded that, as in all disease, different patients react to pain differently, and that psychic disturbances such as fright, excitement and strong emotions, can initiate symptoms of precordial distress (Chapter XIV). The etiological factor in psychic disturbances naturally varies, but fundamentally it consists in abnormal excitation starting from the cerebral centers; the effect upon the circulation, however, may be as disasterous and as pain-producing as the physical factors above enumerated.

There is perhaps no single subject in the realm of cardio-vascular disease of greater clinical interest and importance than that of coronary disease, a common cause of precordial distress. Diagnosis is often difficult because of the many clinical complexes that coronary disease can produce. The difficulty is unfortunately enhanced by the many terms and types introduced by the older writers. Thus, Huchard has divided angina pectoris into true and false. Osler describes four types. False angina pectoris, angina sine dolore, angina vasomotoria, angina vera are terms handed down to us from older clinicians, and now are bandied about with no clear etiological or pathological picture in mind. If one excepts "angina sine dolore" (angina without pain), the common outstanding characteristic, whatever the nomenclature followed, is pain. It therefore seems more logical to retain cardiac, or better still, precordial pains as the predominent feature, and to specify the etiological factor which seems mainly or entirely concerned as the direct cause of the pain. Nothing can conduce to a clearer conception of this involved subject than a searching insight into the pathological possibilities of coronary disease; a good anatomical picture of coronary distribution; a general knowledge of the rich ganglionic nerve supply and plexuses surrounding the heart and

aorta; the correlation and connection between the nerve supply of the heart and stomach; blood pressure estimation; a careful clinical history and urinary examination: These, the handy armamentarium of the thorough clinician, are the fundamental factors upon which diagnosis must be based. One should add, wherever possible, all the more modern methods of refined examination such as electrocardiographic and X-ray examinations, chemical examination of the blood, phenolsulphonephthalein output, Wassermann examination. But often in consultation and general practice, one is suddenly confronted with an apparently unexpected attack of precordial pain of coronary origin, a bolt out of a clear sky, and one must arrive at a definite diagnosis, give an opinion and advise rational therapy with only ordinary clinical methods as our aids.

Before dealing with the pains of coronary disease in detail, it will be advisable to call attention to some general salient features.

1. **Occlusion of Smaller Coronary Branches.**—Regarding the coronaries themselves, they are no longer regarded as end arteries (Chapter I); there are numerous communications, especially deep intermuscular branches, by means of which collateral circulation can be maintained. As an obvicus consequence of this collateral circulation, partial occlusion of a larger coronary branch by embolism or thrombosis, or complete occlusion of a smaller branch need not *necessarily* completely cut off cardiac function of the affected part of the heart, for after a varying interval, collateral circulation may, at least for all functional purposes, restore the blood supply, and with it the vitality of the affected musculature. I emphasize the word "necessarily" because even a little thought will show that there are many factors involved in an assumed restoration of blood supply and function. One can readily conceive, for example, that a large embolus which completely and suddenly occludes a large coronary branch will immediately cause major and extremely serious symptoms with probable death. On the other hand, a cause operating gradually in a smaller coronary vessel—for example slow thrombus formation from chronic or subacute arteritis—will not produce sudden myocardial necrosis and will probably allow sufficient time for collateral blood supply, with a more or less complete restoration of cardiac function, despite the later formation of scar tissue at the thrombosed area. Again, sudden sharp occlusions of the main coronaries, by attacking and affecting large myocardial areas, are often accompanied by pericarditis. As will be later shown, this is of great diagnostic importance.

It is known that patients who have recovered from cardiac pains have occasionally shown myocardial scars and other changes resulting from old infarcts. Huchard, in a summary of 145 necropsies of coronary disease with cardiac pains, found five due to embolism of the artery. Recently, several cases with necropsy reports of embolic infarcts of the main coronaries have been described; the patients died within a few days or hours with symptoms of intense precordial distress. I have observed several cases with intermittent pains lasting days or weeks, in which the symptoms were possibly

caused by infarcts or emboli of the smaller coronary branches. Curschmann in 1891 first described this condition; he reported three cases with necropsies. Two patients died some years after the onset of symptoms. Both showed localized myocarditis. In one, there was an aneurismal dilatation confined to one sclerosed arteriole; in the other there was an obliterated coronary branch of the third order. The third patient, in whom the condition was correctly diagnosed, died suddenly; an embolus was found in a coronary branch of the second order.

In addition to actual plugging of the vessels, there is again the much disputed question of coronary spasm. From experimental observations, we know that coronary spasm can occur. Upon clinical grounds, I believe that coronary spasm can occur in comparatively healthy vessels which are the subject of excessive nerve excitation. For example, I consider coronary spasm the chief cause of precordial pains in so-called "smoker's heart" (Chapter XXVI).

**2. Pain.**—So far as known, there are no nerves of sensation in the heart. Through the fundamental work of Sherrington, Head, and later, of Mackenzie, it is known that a viscus, though not possessed of nerves of sensibility, may, when irritated, excite the corresponding visceral segment of the spinal cord; the latter then sends abnormal centrifugal impulses to the muscles, glands, etc., which in the skin give rise to abnormal sensations usually felt and denoted as pain. In cardiac disease the area ordinarily affected is the precordium. Depending upon the nerves involved, upon the intensity of the irritation, or possibly upon the irradiation of centripetal impulses to other spinal segments, pain may spread to the entire chest, to both arms (especially to the left), to the fingers, neck, head, the interscapular region, the epigastrium, the abdomen and even the thighs. Besides the precordium and left shoulder, the epigastrium is the favorite site for referred pains, a fact which often causes erroneous diagnoses, and mistakenly directs the therapy to the stomach. In severe cases, pain is usually sharp, lancinating or agonizing in character, and is combined with the oft-described feeling of impending death. In milder cases, it is dull and aching in character, or there may be merely a feeling of oppression of the chest. Head's zones of hyperasthesia are sometimes present, usually over the precordium, more rarely in the epigastrium. Occasionally pressure even of wearing apparel, produces severe pain. The sensitiveness may be confined to the underlying intercostal muscles; deep pressure alone then elicits pain. It is sometimes possible in a general way to judge of the effect of therapy and of the progress of the disease by the amount of pain elicited upon superficial or deep pressure. When progress is favorable, sensitiveness to pressure is diminished.

While pain is the most common and indeed a cardinal symptom, the patient may become so overwhelmed by other phases of the disease, for example, by vomiting, dyspnœa and pulmonary edema, that pain as a symptom may be either evanescent or of but minor importance.

3. **Gastric Symptoms.**—Vomiting is extremely common, indeed so much so that in every case of vomiting in an older individual, especially when combined with dyspnœa and with no evidence of any abdominal disease, a coronary infarct should be suspected. The gastric symptoms are usually attributed to a dilated stomach or to congested gastric mucosa, and the dyspnœa to the embarrassed heart action from pressure of a dilated stomach against the diaphragm and heart. My opinion is that, in the vast majority of cases, the cause of gastric symptoms is due to reflex excitation of the vagus nerve supply of the stomach. Indeed the gastric symptoms may mask the actual cardiac picture for years. For example, one of my patients had dyspeptic symptoms resembling severe hyperacidity for years. The chief hints of cardiac damage were some dyspnœa when out of doors, occasional severe pain in the left chest, and a low blood pressure. He finally died of a coronary occlusion attack. In this connection it is important to discuss the frequency of epigastric pains and of occasional epigastric Head's zones found in heart disease. The former are usually ascribed to an enlarged liver or to congested gastric mucous membrane. However, the pains are present when the liver is not enlarged, and necropsy reports show that such patients often had no congestion or disease of the gastric mucosa. Besides, we possess no data which definitely correlate such presumed congestion with pain. From clinical manifestations, from disappearance of epigastric tenderness with disappearance of decompensation, and from the intimate correlation of the nerve supply of stomach and heart, it seems probable that this type of pain is chiefly, if not entirely, the result of referred nerve excitation from cardiac disease.

Further, it should be remarked that the pains of cardiac disease are especially apt to be evoked in those who have actual gastro-intestinal or gall bladder disease. This is especially true of middle aged or older individuals with cardio-sclerotic or coronary disease. I do not believe that in the vast majority of cases, assumed intestinal toxemias play any role in the causation of these precordial pains. The etiological factor seems to be reflex excitation of cardiac innervation originating from disturbances of the gastric, intestinal or gall bladder nerve supply.

4. **Arrhythmias.**—Cardiac irregularities frequently accompany attacks of coronary occlusion; the more severe the attacks, the longer the irregularity is apt to remain. Complete irregularity of pulse and heart action—auricular fibrillation—is the usual type of arrhythmia. Extrasystoles are also common. What I have always considered of special prognostic importance is the occurrence of frequent and apparently unaccountable attacks of arrhythmias: Auricular fibrillation, extrasystoles and paroxysmal tachycardia. Occurring with dyspnœa and accompanied by little or no pain in patients with cardiosclerosis, they usually presage an attack of coronary occlusion which may end in sudden death. Such instances will be cited later.

5. **Acute Pulmonary Edema.**—This very frequently accompanies the precordial attacks of coronary disease. It varies in degree, at times almost immediately causing the patient's death. It is the chief alarming symptom in major attacks. While its cause is still speculative (Chapter XIV), it seems to be directly due to suddenly impaired and tremendously weakened cardiac function, in consequence of which there is pulmonary congestion as the first phase, and edema as its second and final phase. Often pulmonary edema is the symptom which requires immediate therapy.

6. **Transient Nephritis and Dependent Edema.**—In some attacks of coronary occlusion, especially of the larger branches or main vessels, there is often a heavy trace or a cloud of albumen in the urine with many hyaline and granular casts. Hypertension, if previously high, usually decreases somewhat. The urinary condition seems due to general circulatory collapse following coronary failure, with consequent renal congestion. When the circulation improves, the edema and signs of nephritis rapidly clear up. If the coronary attack was not sufficiently typical to make a definite diagnosis from the usual signs and symptoms—precordial pains, pulmonary edema, arrhythmias, dyspnœa—one may venture the diagnosis upon the type of nephritis and edema above described. The treatment is that of the primary condition.

7. **Gastro-intestinal Paresis.**—I have encountered this condition in several cases of coronary attacks when circulatory failure went from bad to worse. The patients are usually in coma or semi-coma. They regurgitate or vomit coffee-ground material; the abdomen is rotund, tympanitic, somewhat doughy. There is no local tenderness. The condition seems due to venous overfilling of the splanchnic reserve areas from cardiac failure, with consequent insufficient oxygenation of the gastro-intestinal tract. Besides cardiac stimulation, gastric lavage, pituritin or adrenalin are especially indicated.

8. Mottling of the extremities or actual venous thrombosis is sometimes observed in the terminal stages of coronary occlusion. The cause is venous or capillary stasis from circulatory collapse.

I have sketched at some length these important and prominent general aspects of the subject in order to clarify the problem of diagnosis, differential diagnosis, prognosis and therapy.

The commoner causes of precordial pains due to cardio-vascular disease, may be tabulated as follows:

A. Organic cardio-vascular disease:

1. (*a*) Hypertensive cardio-vascular disease with myocardial insufficiency. (*b*) Hypertension with myocardial insufficiency and labile vasomotor mechanism. (*c*) Uremic group.
2. Myocardial insufficiency without hypertension.
3. Acute rheumatic endocarditis and rheumatic endocarditic exacerbations.
4. Endo-myocardial disease with general circulatory failure.
5. Embolic infarcts in the main coronaries and their branches.

6. Cardiac syphilis.
7. Premature arterio-sclerosis and cardio-sclerosis.
8. Senile arterio-sclerosis and cardio-sclerosis.
9. Sacculated aneurysm.

1. (*a*) **Hypertensive Cardio-vascular Disease with Myocardial Insufficiency.**—The patients of this group probably represent the most frequent sufferers from precordial pains. The cause of hypertension has been ascribed by some to an increase of epinephrin in the blood, though careful experiments by Janeway and Park have not justified this assumption. Recent studies of blood metabolism have shown that chronic nephritis with hypertension is often accompanied by an increased amount of non-protein nitrogen in the blood, and by a diminution of blood alkalinity, (Chapter XXI)—facts which may in the future offer promising fields for therapy. The chief cardiac changes are ventricular hypertrophy, usually left, but sometimes also right; patchy, fibrous myocarditis; thickened aortic and mitral cusps; lime deposits on the first portion of the aorta; and atheroma and thickening of both coronaries. It must be remembered, however, that patients with similarly diseased hearts may have little or no precordial distress.

A brief case description will serve to fix the type: A systolic blood pressure of 190 m.m.; a rough first and a sharply accentuated and bell-like second sound at the right base; evidence of marked left ventricular hypertrophy with a heaving apical impulse; urine with or without albumin or casts; very slight pretibial edema; dyspnœa on exertion or appearing suddenly at night; nycturia. The pains are usually dull, most marked in the precordium, and radiate to the neck and arms.

Almost frantic therapeutic efforts are made, as a rule, to reduce the high blood pressure, while the fact that it is often a conservative, compensatory process seems to be entirely ignored. A mere enumeration of the long list of remedies is sufficient proof of their inadequacy. Vaso-dilators (nitroglycerin, amyl nitrite, erythrol tetranitrate), hot, Nauheim and oxygen baths, violet ray, diathermy and electric light baths are the most popular. There are conflicting and contradictory reports regarding all. In some cases, I studied the effects of hypodermic injections of 1 per cent. solutions of nitroglycerin in doses up to one tenth of a grain, there times a day; they had no effect upon the symptoms and caused but an occasional temporary reduction of the blood pressure. The physical changes in the coronaries probably account for the futility of vasodilators to regulate the impoverished cardiac circulation and thus relieve the resultant precordial symptoms. While theories such as cardiac spasm and anemia have had vogue as the causes of the pain, it seems more probable that the underlying factor is nutritional cardiac disturbance either from inadequate coronary circulation or, possibly, from toxic products flowing in the general circulation. Acute violent pains call for morphine. Pearls of nitrite of amyl may also be of aid. With milder symptoms, nitroglycerin occasionally gives some relief, especially if combined with atropine

sulphate. Aside from temporary therapeutic measures, I have placed main reliance on digitalis, either the tincture in 15 drop doses, or digitan tablets three times a day. The drug should be given whether the auricles fibrillate or the pulse is rhythmic, and should be stopped when pains and other symptoms improve, and then continued in smaller doses for a long period. A curb should be put on the patient's physical activity. Mental excitement and stress should be avoided. The theoretical objection that digitalis may perhaps cause or increase coronary spasm has not been verified by clinical evidence in cases in which the drug has been given for months. In only one of my cases were precordial pains increased by digitalis, although even here there had been general improvement for several months.

The importance of treatment of edema and other manifestations of cardio-vascular disease lies in their fundamental association with precordial pains. This is illustrated in the following case:

A vigorous woman of 43, never pregnant, complained for three years of dyspnœa on walking, and of stabbing precordial pains radiating to the left shoulder. The Wassermann blood reaction was negative. The urine contained a trace of albumin and, occasionally, hyaline casts. There was slight tibial edema. Physical examination revealed typical signs of aortitis. The orthodiascopic tracing showed an enlarged left ventricle and a dilated aortic arch. The precordial area was tender to deep pressure. For the first few months she was put on digitalis, theobromin sodium salicylate, and occasional Karrell days (Chapter XX). Within two months the cardiac pains and pretibal edema disappeared. Later, when she became remiss about medication and diet, dyspnœa and pain recurred. Under stricter surveillence and the same medication, combined with absolute rest at home on the Karrell days, twice weekly, she again slowly improved. This plan of treatment has been followed for several years. The blood pressure is still high; data revealed by physical and roentgen examinations are the same, but there is very marked clinical improvement; precordial pains and edema have entirely, and the dyspnœa almost entirely, disappeared.

(*b*) **Hypertension and Myocardial Insufficiency with Labile Vaso-motor Mechanism.**—In a smaller group of cases, in which the highest systolic blood pressure was around 180 m.m., with marked diurnal variations of as much as 30 m.m., precordial pains following exercise were the main symptoms. Nephritis was apparently not the main or the only cause of hypertension; emphysema and myocarditis were the chief pathologic conditions. Experimental subcutaneous injections of nitroglycerin in doses of one fiftieth grain three times a day had a marked temporary effect on the blood pressure and, usually, on the symptoms; in one instance, the injections were regularly accompanied by sudden relief of precordial pains, to be followed by giddiness. Such cases apparently represent examples of disturbed labile vaso-motor mechanism rather than hypertension due to marked vascular disease alone. Nitroglycerin or other vasodilators given at the onset of pain are apt to be

followed by great relief. Digitalis, though useful, is not as beneficial as in Group I. This is the group in which vaso-dilators are apt to be followed by beneficial results, apparently because coronary spasm plays an important role in the causation of symptoms.

(*c*) **Uremic Group.**—Headache, nausea, vomiting, varying grades of anemia, attacks of paroxysmal dyspnœa and of precordial distress, high systolic and, especially high diastolic pressures, nocturnal polyuria and changes in the retina, are the cardinal signs and symptoms. Kidney test meals, as advocated by Schlayer and others, usually show decreased salt, nitrogen and water elimination; and the blood shows retained non-protein nitrogen. The facial appearance in typical instances is characteristically pallid or ashy grey.

The precordial pains are not relieved by nitroglycerin, digitalis or theobromin sodium salicylate. They are apparently caused by toxic products flowing in the general circulation and affecting cardiac nutrition. Dietetic measures, especially a diet low in protein and rich in carbohydrates, are of most value. When renal tests show no water retention, ingestion of large quantities of fluids is sometimes beneficial. For the relief of pain, occasional doses of morphine are necessary. On the theory of tissue acidosis, I tried the intravenous injection of 500 c.c. of a 5 per cent. bicarbonate solution in two cases (Chapter XXI). In one, there was marked relief of the precordial pains and other symptoms; in the other, the pain was relieved for a few hours only. Glucose solutions given by the Murphy drip or even intravenously may be of value.

2. **Myocardial Insufficiency without Hypertension.**—The main complaint of these patients, usually men of sedentary habits between the ages of 50 and 60, is slight precordial pain following exertion. The patients are well preserved. The urine is normal or may contain a slight trace of albumin with a few casts. Physical examination reveals a slight impurity of the first sound at the apex, and a soft systolic murmur at the base. There is no evidence of severe cardio-vascular disease. The systolic blood pressure is around 160 m.m., rarely much higher; there is no edema. The orthodiascopic tracing usually shows a slightly dilated aortic arch with the left ventricle lying broad and flat on the diaphragm; it is impossible to state if this ventricular contour is due to flabbiness or to hypertrophy (Chapter XII). It is interesting to note that these patients commonly give a history of having had painful gastric attacks in previous years, with symptoms pointing to gastric or duodenal ulcer. Excellent results have followed the use of digitalis, given at first in large, and then in moderate doses, and continued intermittently for weeks or months. Small doses of atropine were sometimes added. At the beginning the patient's activity was somewhat restricted; later, moderate exercise—golf or walking—was advised. Acidulous food and drink were interdicted, and some light form of nourishment between meals was prescribed. The following is an illustrative case:

Male, age 60, for years has been careful about his diet, particularly in the avoidance of acids, because indiscretions were followed by epigastric pains. For several months he has complained of precordial, knife-like, paroxysmal pains on walking. Examination showed a well nourished man; there was no edema; the urine contained no albumin or casts; the average systolic blood pressure was 160 m.m. The orthodiascopic tracing revealed a somewhat enlarged left ventricle, the aortic outline was slightly broader than normal. There was an impure first sound at the apex and at the right base. There was no dyspnœa or decompensation. One digitan tablet was given, at first three times daily, and then continued in smaller doses for several months. Atropine sulphate, $\frac{1}{150}$ of a grain, three times a day, was also prescribed. The patient is now doing his regular work, has taken ocean baths during the summer, and has had no recurrence of precordial pains.

3. **Acute Rheumatic Endocarditis and Rheumatic Endocarditic Exacerbations.**—These cases usually occur in young persons with definite rheumatic histories, with mild tachycardia, with no dyspnœa, and with marked auscultatory evidence of valvular disease (usually mitral stenosis). Slight irregular rises of temperature are common. The patients complain not only of the subjective feeling of palpitation, but also of "sticking" pains localized in the region of the heart. There are usually precordial Head's zones of hyperasthesia. The cause of the rapid heart action and of the precordial pains, even in the absence of abnormal temperature and other rheumatic manifestations, appears to lie in the irritative effect of fresh exacerbations of endocarditis. The best medication is sodium salicylate given in full therapeutic doses. My routine has been 15 grains hourly until six doses have been given or until tinnitus occurs; the dose is then decreased. Bromides in moderate amounts, and ice bags to the precordium, are also helpful. Moderate or absolute rest in bed may be necessary for some time.

The following are typical instances:

Female, unmarried, aged 18, under observation three years, gave a typical rheumatic history. There is a marked double aortic lesion and a tremendously hypertrophied heart. The Wassermann reaction and frequent blood cultures were negative. The systolic blood pressure was 180 m.m., the diastolic, 20 m.m. For months she has had many attacks of moderate irregular fever. On several occasions these were initiated by sharp precordial pains followed by tachycardia or auricular fibrillation, convulsive twitchings and tremors, and by loss of consciousness lasting several days. Latterly these attacks have become more severe and are followed by well localized Head's zones.

Female, unmarried, aged 20, gave a history of continued attacks of rheumatism and "heart trouble." During the last few months she had two attacks similar to but not quite as severe as that with which she entered the hospital. There were continued agonizing pains and exquisite tenderness, even to the slightest touch, over the precordium, and radiating pains to the

shoulder and forearm. The temperature was 104°. A mitral regurgitant lesion was present. There was no edema. Breathing was frequent and shallow, apparently in the attempt to keep the chest at rest. After one week, high temperature, sharp pain, and rapid breathing disappeared; the patient felt comfortable, but precordial tenderness on firm pressure was still present when she left the hospital.

Male, aged 40, with a double mitral lesion, gave a history of having felt well until three months prior to hospital admission. He then had an attack of mania (?) lasting three days. Subsequently pneumonia and pleurisy developed, accompanied by chills and high fever and by attacks of paroxysmal auricular fibrillation. After several cultures, a non-hemolytic streptococcus was isolated from the blood. During several months of hospital observation there was a constant sensitive area near the cardiac apex.

The characteristic of these three cases was not only the rise of temperature and evidences of active endocarditis, but also the progressive tendency of the disease, and the presence of local, tender precordial areas. Though these manifestations may have been due to the endocarditis alone, the distinct localized sensitive areas, and the clinical course, make it probable that, in addition, acute focal myocarditis was present. From clinical and physical signs, and from the progressive and probable bacterial nature of the endocarditis, it seems fair to assume that such focal myocardial changes originated in embolic infarcts of the smaller coronary branches, accidents not necessarily incompatible with life.

Necropsy findings will of course be necessary to establish the diagnosis in such cases.

Two illustrative cases are given to show the diagnostic importance of dry pericarditis, as well as to indicate the difficulties of diagnosis.

Mr. K. aged 80, a man of powerful physique, had always been well until two days before I saw him. He was then attacked by severe precordial pains which were almost continuous. Upon examination, there were found hypertension, thickened and tortuous arteries, loud systolic murmur over the mitral, a definite patch of dry pericarditis extending over the mitral area. The patient died of pulmonary edema after two days.

Mr. S, aged 53, had chronic nephritis, hypertension and left ventricular hypertrophy for several years. Of late months he suffered from almost constant dyspnœa, from occasional slight attacks of pulmonary edema and from attacks of precordial pains. In at least three such attacks, as accompanying features, I was definitely able to localize areas of dry pericarditis with typical friction rubs over the lower precordium. With the recession of these attacks of pain (which I ascribed to thrombosis in the smaller coronaries) the physical signs of dry pericarditis also disappeared. As I visualize the pathological process, each attack of coronary occlusion was accompanied by a patch of fibrinous exudate over the freshly invaded myocardium; the exudation again receeded either as the result of the estab-

lishment of collateral circulation or because the acute inflammatory disturbances had run their course.

4. **Endo-myocardial Disease with General Circulatory Failure.**—The fundamental cause of the pains in apparently nutritional disturbances from local circulatory failure in the heart itself. Precordial Head's zones or intercostal muscle tenderness are often present. The therapy for the relief of pain is the same as for decompensation. This ordinarily means vigorous and long-continued digitalis medication, and, for the relief of edema, theobromin and the dietetic regimen already outlined. If compensation is restored, cardiac pains and Head's zones disappear.

5. **Cardiac Syphilis** (Chapter XVI) is an extremely frequent cause of precordial pain. It is usually substernal, dull, boring and aching; it may, however, have the distribution and characteristics of the types already described. One can probably ascribe the frequency of these substernal pains to the almost invariable presence of syphilitic aortitis and periaortitis with consequent aortic dilatation; to the fact that the root of aorta is surrounded by rich ganglionic and nerve plexuses; and to the varying degrees of aortic dilatability and pressure. Head's zones are comparatively rare. There are apt to be acute attacks of precordial pains if the coronaries are also involved. Such attacks are frequently accompanied by decompensation. Therapy consists in the treatment of the underlying disease (Chapter XVI). Although salvarsan was originally considered contraindicated in cardiac syphilis, abundant experience has since shown that, given first in smaller, later in larger doses, salvarsan combined with the usual mixed treatment is of great and definite value. The best routine method of administration is 0.2 grm. of salvarsan or arsphenamin injected intravenously every week until 0.6 grm. is given; later, full doses may be given, the frequency depending on the cardiac condition. Salvarsan sometimes benefits and controls the pains immediately, apparently because of reduction in the syphilitic inflammatory exacerbations. Where the pathologic process in the aorta, the coronaries or in the myocardium has reached an extreme degree, salvarsan or any other treatment can be of little or no avail. Cardiac failure, if present, should receive its appropriate treatment, that is, digitalis, and if necessary, theobromin sodium salicylate.

6. **Premature Arterio-sclerosis.**—This comprises a rare group found in young adults. Persistent precordial distress is often present for months. Gastric symptoms similar to those of hyperacidity occasionally dominate the clinical picture. Physical examination may give no hint of the severity of the pathological process attacking the entire cardio-vascular system, a process finally resulting in extreme changes throughout the aorta, coronaries, arterioles and endocardium. Indeed, such hearts may be pathologically identical with those of persons dying with senile cardiac changes. For months there may be no decompensation in the ordinary sense. The urine may be normal, the blood pressure not high, the heart sounds somewhat

distant, and the only indication of severe cardiac disease, besides the precordial pains, may be the presence of an arrhythmia, usually marked sinus arrhythmia or extrasystoles. Other occasional symptoms are dyspnœa and nocturnal attacks of precordial distress.

An illustrative case with necropsy report follows:

Male, aged 42, of athletic build, had been a heavy smoker. For four years he had suffered from gastric symptoms resembling hyperacidity; belching was particularly prominent. The gastric contents showed high values for free hydrochloric acid. He had frequent attacks of vasomotor disturbances; numbness, coldness and pallor of the hands. It was only late in the disease, when an attack of hemiplegia occurred, that attention was focused on the possibility of a generalized arterio-sclerosis as the underlying disease. Although several Wassermann tests were negative, salvarsan and mixed treatment were given; these were without effect. At necropsy, marked thickening and calcareous deposits were found on all the cardiac valves; the coronaries and their branches showed extreme thickening; the myocardium presented many fibrous patches; the entire heart was somewhat enlarged. Spirochetes were not found in the aortic tissue. The heart accurately resembled that of a very old person suffering for many years from severe generalized arterio-sclerosis and cardio-sclerosis.

The cause of such presenile sclerosis is still undetermined. It is possible that unknown infections and toxemias are etiologic factors. In one of my patients the disease originated within two years of an obscure pulmonary infection of several months duration. For the present, one can only state that, from some unknown cause, the elastic tubing comprising the vascular system becomes prematurely defective and diseased. Except digitalis for temporary relief of dyspnœa, treatment is of no avail.

**7. Senile Arterio- and Cardio-sclerosis.**—The clinical picture is usually clear. All the palpable arteries are tortuous and thickened; the blood pressure is normal or not very high; the heart, somewhat enlarged; the systolic sounds at the apex and right base are impure; the second sound at the right base is sometimes accentuated. Precordial pains usually occur with exertion; although sometimes intense, they are more often of mild character. Apparently they basically depend on coronary sclerosis. Because of the advanced pathologic changes, the patients can rarely be benefited by medication. Digitalis and theobromin sodium salicylate sometimes help, the amount of relief seemingly depending upon the degree of healthy tissue still remaining to react to medication. The patients usually require prolonged periods of comparative rest.

**8. Sacculated Aneurisms** (Chapter XVI).—In a general way, the precordial pains depend on the size and position of the aneurism. If large, the pains are those due to pressure on the surrounding structures (intercostal nerves, ribs, etc.). If small, and involving the first portion or arch of the aorta, the symptoms are similar in character and etiology to those described

under cardiac syphilis (Group VI). The therapy is also the same as that there indicated. My observation with wiring and the electric current treatment according to the method of Lusk is limited to two cases. In one, after some weeks, there occurred small frequent external hemorrhages due to perforation of the sac by the end of the wire; the patient finally died of anemia. In the other, the patient did not improve during his stay in the hospital; he died suddenly some weeks later; a necropsy was not obtained.

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## CHAPTER XXIV

## PRECORDIAL PAINS OF EXTRA-CARDIAC ORIGIN

Precordial pains are so frequently the result of disease and disturbances outside the heart, and are then so often attributed to some form of "heart trouble" that it is important to know the commoner extra-cardiac causes of precordial pains, and to indicate how they may be distinguished from the precordial pains of organic cardiac disease.

**Precordial Pains Due to Tabagism.**—For reasons already given (Chapter V), I have classed the "tobacco heart" with the inorganic cardio-vascular diseases. The precordial pains may be dull and aching, or sharp and radiating; occasionally, the first premonition is very sharp lancinating precordial pains radiating to the left shoulder and forearm, and accompanied by unconsciousness. As an instance, a healthy man of 40, a heavy smoker, while running for a train, was suddenly attacked by terrific precordial pain followed by unconsciousness for one hour. The cardio-vascular examination revealed nothing abnormal. For weeks after the attack, any slight movement, even turning in bed, brought on pains. The patient was finally able to be about and resume his usual occupation. At present, six years after the first fainting spell, he feels quite well. He again smokes, but moderately.

The most frequent of the arrhythmias in smokers are extrasystoles, usually auricular. Other arrhythmias, however, are occasionally encountered. Thus I have seen two cases of sino-auricular block (Chapter X), one of auricular flutter and one of auricular fibrillation.

Most tobacco pains and arrhythmias cease when smoking is stopped. Occasionally both recur intermittently for years. In such instances nitroglycerin given regularly or during pain, is of benefit. The bromides are of value in helping to control persistent arrhythmias.

**Gastric Disturbances.**—Patients with gastric disturbances, particularly those in whom epigastric distress is marked, are apt to suffer from referred precordial pains. The clinical picture is that of hyperacidity, or of gastric or duodenal ulcer. Belching, loud and explosive in character, is usually a prominent symptom. The precordial pains are commonly sharp, neuralgic, fleeting, and inconstant in character; their distribution is usually along the fourth and fifth left intercostal spaces. Pain referred to the left shoulder is rare. Corresponding to the anterior distribution there may be a similar area posteriorly. A precordial Head's zone is unusual; its area of distribution, inconstant. There may be a close association between gastric symptoms and precordial pains, particularly when "hunger pains" and belching and marked. There is no decompensation or dyspnœa. When gastric symptoms

are stormy, extrasystoles may occur. Treatment is naturally directed to the underlying disease. I have found most benefit from atropine given in full physiologic doses three times a day before meals, and from an ant-acid powder containing equal parts of sodium bicarbonate, magnesium usta and oleosaccharated peppermint powder in half teaspoonful doses after meals. Appropriate bland diet given frequently and in small quantities is important. As already noted, gastric symptoms also accompany cardiac disease, so that the correlation and study of all the clinical data are necessary for a correct diagnosis. Unless extrasystoles are subjectively annoying, they require no medication; otherwise they may be partly controlled by bromides or luminal (Chapter XX).

**Cardiospasm with Temporary Hypertension and Precordial Pains.**—The usual impression gained from the literature is that cardiospasm is constant, that it is accompanied by painful deglutition when food reaches the cardia, and that esophageal dilatation is frequent and often extreme. Only recently was I able objectively to verify my clinical impression that many *mild* cases of cardiospasm occur without any of the marked symptoms and signs above enumerated. The case was that of an unmarried girl of twenty-two whose appendix had been removed some months previously for dyspeptic symptoms apparently originating in a diseased appendix. Some dyspeptic symptoms still remained: These were chiefly sudden distention, gas and eructations after meals. Fluoroscopy of the stomach with a barium meal showed marked pyloric spasticity, so that only after long continued vigorous massage of the stomach was some of the barium mass seen to enter and fill out the duodenal cap. Of most interest to me was the fact that an added drink of barium in milk showed that it remained in the lower esophagus near the cardia, that slight anti-peristalsis took place and that when the barium mass finally entered the stomach, the cardia and lower esophagus formed one long spastic elongated tube filled with the barium. This introduction is of especial importance for what follows.

In a review of patients who sought relief for precordial pains, I was impressed by the comparative frequency of a group who, in addition to the pains, gave clinical evidence of mild cardiospasm. They sometimes complained of slight epigastric distress at the end of the act of swallowing: Their appetites were good; their only gastric complaint was a feeling of distention and a desire to belch after meals; if they could belch freely they were relieved. The only definite findings on abdominal examination was a varying degree of deep seated tenderness under the xiphoid, best elicited by pressing the finger in the epigastrium, not however toward the spinal column, but upward and under the xiphoid. The only definite abnormal physical finding upon thorough cardio-vascular examination was moderate hypertension, usually between 160 and 170. The heart, urine, phenolsulphophthalein, orthodiascopic and electrocardiographic examinations presented no deviations from the normal. There was no dyspnœa upon exertion, no edema of

the legs. In addition to these symptoms, patients complained of precordial pains on walking, especially when first leaving their homes and encountering the fresh air; this was apparently due to vasomotor instability, a condition also found in precordial pains of organic organs (Chapter XXIII). The hypertension, the precordial pains, together with the fact that the individuals affected are usually of middle age make a clinical picture, which symptomatically resembles actual coronary disease, hence the importance of making a differential diagnosis.

Illustrative cases are as follows:

E. N. O., male, aged 50, was always a rapid irregular eater. For years he suffered from explosive belching and slight epigastric distress after eating. He never vomited. His appetite is always good. He is a moderate smoker. For one year he complained of precordial pains radiating to the left arm and hand, accompanied by tingling sensations in the fingers. At times the pains were so severe that he had to stop walking. He also had slight dizzy spells during the last six months. As the result of a thorough cardiovascular and general clinical examination, including orthodiascopy and electrocardiography, the only definite abnormal findings are slight deep seated sensitiveness under the xiphoid, and moderate hypertension: The systolic blood pressure was 160, the diastolic 100. The diagnosis of cardiospasm was made, the patient was put upon a bland ant-acid diet and regular meals. Thorough chewing of food was insisted upon. A good prognosis was given. .An ant-acid powder to be taken after meals, and small doses of atropine before meals were prescribed. Within one week after therapy directed entirely to the stomach, the hypertension disappeared. The pains disappeared more slowly. Several weeks after the original examination, the patient felt entirely well, the systolic blood pressure was 120, the pains had not recurred, the patient was able to walk and to attend to his business without precordial discomfort.

S. L. male, lawyer, aged 39, always stout, had been under treatment for hypertension and precordial pains for many months. The blood pressure varied from 160 to 180 systolic. He had been under a vigorous protein-free dietary regime during the same period. Inquiry elicited that the patient suffered from flatulence for years, that he was exceedingly busy and under nerve tension in his office work, that he ate very irregularly but voraciously, that he smoked 30 cigarettes daily. A fortnight before I saw him, he had an attack of sudden severe substernal pain after a heavy meal: The pain was aggravated by walking. The pains now radiate to both arms and occasionally even to the legs. There was no history of tonsillitis or rheumatism, there was no dyspnœa after exertion. The Wassermann blood test was negative: The urine, normal. The only abnormal finding upon cardio-vascular examination, including orthodiascopy and electrocardiography, was slight hypertension. Abdominal examination revealed an obese abdomen; there was a small definite area of deep-seated sensitiveness

under the xiphoid. The amount of smoking was reduced. Regular and slowly eaten meals were insisted upon; a bland ant-acid diet was ordered: Tablets containing 1/150 grains of atropine sulphate and 1/150 of a grain of nitroglycerin before meals, and an ant-acid powder of equal parts of magnesium usta, bicarbonate of soda and oleosacchorated peppermint powder after meals was prescribed. The pains and hypertension gradually disappeared, epigastric sensitiveness became less. During the summer the patient was able to swim, row and climb hills without any discomfort. I have seen the patient several times since then; the blood pressure has remained normal.

I believe that in this group of cardio-spastic patients with hypertension, organic cardio-vascular disease whether of the heart, systemic arteries or kidneys, can be excluded because of the negative findings in the circulatory system after careful clinical investigation, and because they yield so readily to therapy directed to the affending organ, the stomach. It is known that the cardiac end of the stomach is richly supplied with nerve structures, mainly branches of the vagus. Upon clinical, physiological and therapeutic grounds it seems to me that the hypertension as well as the precordial and referred pains are in some manner brought about by referred nerve excitation from a hyperirritable spastic cardia. The etiology of the cardiospasm seems quite clear in the type of patients we are dealing with; namely, constant dietetic insults from irregular and improper eating, and from bolting food. The individuals themselves are often not neurotic in the ordinary sense of that term: Indeed they are as apt to be phlegmatic. But once injury to the cardia is established, other reflex areas may be disturbed and produce the clinical syndrome outlined above.

**Precordial Pains from Esophageal Disease and Spasms.**—Carcinoma, syphilis, ulceration and diverticula are the usual organic causes of referred intercostal and precordial pains of esophageal origin. In some cases, organic disease is absent; the symptoms may then be due to esophageal spasm, especially in the region of the cardia. The usual symptoms are an uncomfortable, conscious gulping effort on swallowing solids or fluid, accompanied by sharp pains referred to the lower sternum, and radiating to the precordium or even to the upper and lower extremities. Such symptoms are occasionally mistaken for those due to aortic aneurism; the difficulty in swallowing, to aneurismal pressure on the esophagus.

An illustrative case with therapy follows:

Male, aged 48, stated that for seven years he had "choking sensations" in the larynx and severe substernal pains on attempting to swallow solid food. These pains occasionally radiated to the head, arms and legs. The symptoms became progressively worse. The neurologic and physical examinations revealed nothing abnormal; the fluoroscope showed a normal aorta. Wassermann reactions of the blood and spinal fluid were negative; the stools, urine and gastric contents were normal. Roentgenograms showed a pouch-like dilatation of the middle part of the esophagus. An esophageal bougie was

arrested opposite the middle of the sternum. Finally a small bougie was passed. For purposes of dilatation, bougies of increasing caliber were employed. At first semisolid food was given. Atropine sulphate, $\frac{1}{100}$ grain three times a day was administered. The pains gradually subsided and finally disappeared. The patient was taught to pass a stomach tube; this he did for some weeks after he left the hospital. An examination months later showed that esophageal symptoms and pains had disappeared, and that there had been considerable increase in weight.

**Precordial Pains Accompanying Crises of Acute Pulmonary Affections.**—Such attacks of precordial pain are fairly common with the crises of influenzal bronchitis and pneumonia, especially when sharp critical sweats are present. Head's zones in the left nipple region, and arrhythmias (especially sinus arrhythmias and extrasystoles), are frequent, a combination suggestive at first sight of toxic myocarditis. Symptoms, however, last only a few days; except for pain and arrhythmia, the heart is normal; there is no decompensation or edema. Examinations made months or years later show that the cardio-vascular system has not been damaged.

The following is an example:

A physician, aged 43, never had any prior cardiac complaint. He contracted pharyngeal and bronchial grippe. There was critical defervescence, sharp sweats, an irregular pulse, and sharp, stinging pains in the left breast. The patient feared that he had an infectious myocarditis, and carefully avoided any unnecessary exertion. Examination revealed a small Head's zone confined to the left nipple region, and an arrhythmia which polygraphic and electrocardiographic tracings showed to be due to auricular extrasystoles. Otherwise the cardio-vascular system was normal. The patient was reassured, told to get out of bed, and given atropine sulphate, $\frac{1}{150}$ grain three times a day before meals, and bromides at night. He was sent to the country and advised to exercise as much as he chose. He was soon able to walk several miles daily. Within one week the extrasystoles and pain disappeared, and have not since returned.

While types such as those described are apparently not due to any disease of the myocardium itself, they must be sharply differentiated from the occasional case in which precordial discomfort presumably arises from some mild toxic disturbance of the myocardium coincident with or following the infection. The usual characteristic, distinguishing features are tachycardia; slight dyspnœa; exceptionally, a soft systolic murmur at the apex; and a sense of oppression in the left chest rather than any severe precordial pain. The treatment consists of rest in bed until these acute manifestations disappear.

**Precordial Pains Accompanying Vasomotor Disturbances at the Menopause.**—Women at the climacteric period with marked vasomotor disturbances (flushes, heat flashes, cold extremities, etc.), frequently have persistent precordial pains without evidence of organic disease of the heart

or of neighboring organs. The pains occasionally radiate to the left arm and hand. They are possibly evidence of vasomotor circulatory disturbances in the heart itself. The patients rarely react well to medication. Bromides and small doses of atropine and nitroglycerin are of most value. Ovarian extract (corpus luteum) and extract of suprarenal gland tablets are occasionally helpful. Hurry and excitement should be avoided.

**Precordial "Neuralgias" of Unknown Origin.**—Under this heading is grouped a small number of individuals, usually young and apparently healthy and vigorous, without vicious habits, with no rheumatic manifestations, in whom continued and frequent examinations of the heart and other organs reveal nothing abnormal. Occasionally, sudden explosive belching or singultus occurs. In one of my cases, smoking some years previously might have been an etiological factor. The pains are apparently quite haphazard in their onset and duration, and in my experience defy medication and therapy.

In this group may also be placed females who complain of indefinite chest pains and distress, and who, from other manifestations apparently suffer from a functional disorder of an internal secretory organ or organs (endocrine imbalance). These patients are occasionally relieved by medication aimed at substituting the presumed secretory deficiency. The extract of suprarenal gland is of most benefit.

**Pott's Disease, Mediastinal and Spinal Tumors, Tabes, Intercostal Neuralgia and Myalgia, Pleurisy, Pericarditis.**—These are some of the additional obvious extracardial conditions and diseases producing precordial pain. They require mention in order to complete the subject.

The etiology and types of precordial pains ennumerated and described are admittedly incomplete. Only those frequently encountered and clinically important have been discussed. It must be emphasized that the various groups sometimes overlap and that careful discrimination is necessary in order to distinguish precordial pain of organic cardiac disease from that due to extra-cardiac causes.

## CHAPTER XXV

## ARTERIAL BLOOD PRESSURE—VENOUS PRESSURE—CAPILLARY CIRCULATION

**Physiological Considerations.**—Blood pressure observation has taken its place as a routine method of examination in clinical medicine. Its value and importance in health and disease are based upon the results of experimental physiology, which demonstrated that various factors are concerned in the estimation of blood pressure. These are (1) Cardiac Energy; (2) Elasticity of the Arterial Wall; (3) Peripheral Resistance; (4) Venous System; (5) The Lymphatic System; (6) Volume of the Circulating Blood; (7) Viscosity of the Blood.

1. **Cardiac Energy.**—From the physical standpoint, cardiac work depends on the amount of, and the velocity with which blood is pumped into the arterial system. This in turn largely depends on the amount of venous blood brought to the heart. If the amount of venous return is greatly diminished, as for example, by section of the splanchnics, ventricular filling and, consequently, systolic output are also considerably diminished. The dynamics of the cardiac cycle—systole, diastole and diastasis—as well as the effect of rapid and slow heart action upon the cardiac output have already been described, (Chapter VI). Others factors being equal, an increased volumetric output raises, a decreased output decreases, blood pressure in the aorta. In the normal animal, the pulse rate is retarded by raising arterial pressure, and accelerated by lowering it. This reaction does not usually occur after vagus section; hence it is probably due to action upon the vagus center, partly reflex and partly direct. In animals the centripetal nerve to this center is the depressor which sends terminal filaments to the ventricular musculature and probably also to the aorta (Chapter I). If this nerve is cut or stimulated peripherally, it has no effect upon the heart action or blood pressure. If its central end is stimulated, there follows a marked fall in blood pressure and heart rate. The depressor thus acts as a defensive mechanism against unduly high blood pressure. Its aortic filaments are stimulated by undue distention of that vessel.

2. **Elasticity of the Arterial Wall.**—Because of their elastic distensibility, a large proportion of the force of ventricular systole is stored up in the larger arteries. By their stretching and elastic recoil, arteries act as a reservoir of power after systole has ceased. In this manner the strain of systole upon the cardiac musculature, as well as upon the arterial wall, is considerably diminished. If the arterial tree were a rigid system, the systolic blood pressure, and hence the force of the impact, upon the arteries would be greatly

increased. The diastolic pressure would always be nil, hence there would always be a tremendous pulse pressure, thus again enormously increasing the cardiac load.

**3. Peripheral Resistance.**—This has reference to resistance encountered in the capillaries and end arterioles. The capillaries and veins together act as a reservoir for the blood. They are under the influence of nerve tone, hence any profound factor which decreases this tone will cause an accumulation of blood in the capillary bed and venous reservoir. With other factors unchanged, the blood pressure is increased by greater peripheral resistance and decreased by lessened peripheral resistance. The tension of the normal artery depends on its tonus, which in turn chiefly depends upon the vasomotor mechanism. To a lesser extent, this statement applies to the veins, for they too possess a certain amount of tone. Normal tone is governed by a proper balance between the vasodilator and vasoconstrictor mechanisms, as derived from the regulatory centers in the medulla. There are other subsidiary centers in the spinal cord, demonstrated by the fact that, after destruction of the bulbar center, the arteries gradually recover their tone. In the experimental animal, general vasomotor tone is readily influenced by distant and near reflexes. After stimulation of the depressor nerve the splanchnic vessels become dilated, and blood pressure falls, but stimulation of all the other centripetal nerves raises blood pressure. The abdominal vessels, innervated by the splanchnics, have the greatest effect upon the general blood pressure because they can contain a large amount of blood and are easily influenced reflexly. It is important in this connection to emphasize that reflex lowering or raising the blood pressure does not simultaneously affect vascular areas in the same way.

**4. Venous System.**—It has already been indicated that venous tone exists and that the venous system, especially in the splanchic area, can contain a large part of the entire volume of blood. It is apparent that splanchic overfilling of the venous system would result in a lessened supply of blood to the heart, and hence would decrease the blood pressure. It would correspond to some phases of vasomotor shock (Chapter XXVI) in which patients are assumed "to bleed into their own vessels." On the other hand when the large venous trunks leading to the heart become overdistended as a result of pulmonary or hepatic disease, or of circulatory disturbances, the right heart cannot properly empty itself. This undoubtedly affects venous pressure, although what effect it may have clinically upon the systemic blood pressure is not known.

**5. Lymphatic System.**—The state of filling of the lymphatics is probably a factor in blood pressure by acting as a temporary regulator and distributor of the fluid elements of the blood when, for any reason, the volume of the circulating blood is increased.

**6. Volume of the Circulating Blood.**—The possible content of the arterial and venous systems is much larger than the actual amount of blood found in

the body. The disproportion is equalized by peripheral contraction and by the other factors described; hence normally, blood volume has only a very slight effect upon blood pressure.

**7. Viscosity of the Blood.**—It is evident that the degree of viscosity may have some influence upon blood pressure. Up to the present time, however, there are no clinical or experimental data (except in shock (Chapter XXVI) which have any bearing upon the subject.

All the above factors found in the experimental animal are also present in the human being. Their value and importance vary not only relatively but

FIG. 281.—The Baumanometer—a mercurial sphygmomanometer, illustrating also the application of the instrument for the auscultatory method of reading the pressure. This instrument is accurate, portable, simple in construction and not apt to get out of order. The graduate tube is carefully calibrated.

absolutely from time to time; the blood pressure is the resultant of these variants. Although we posses no methods or instruments by which individual agencies making up the clinical blood pressure can be separately calculated, one should at least attempt to gauge them by careful examination and by such significant data as the thickness of palpable arteries, existence of plethora, state of venous distention, etc.

**Clinical Estimation of Blood Pressure.**—The instruments which are used in the clinical estimation of blood pressure are called sphygmomanometers. The three types are the mercurial, the aneroid and the compressed air. Fundamentally they depend on calculating the power exerted in partial or complete

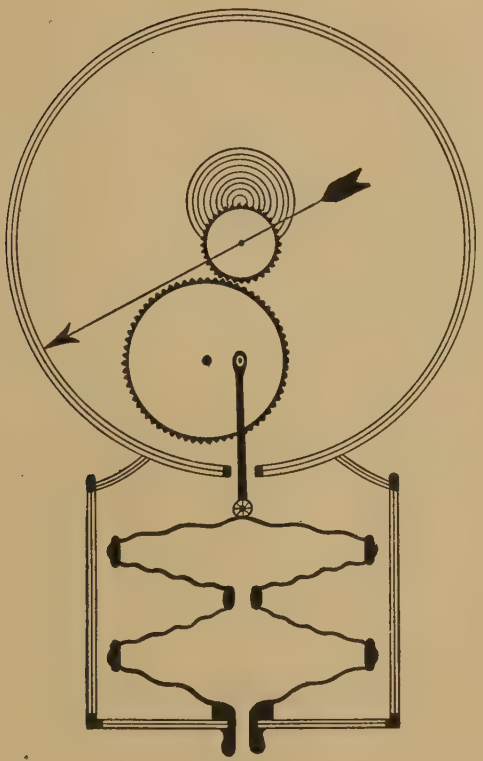

FIG. 282.—Construction diagram of the Tycos Aneroid. (*After Norris.*)

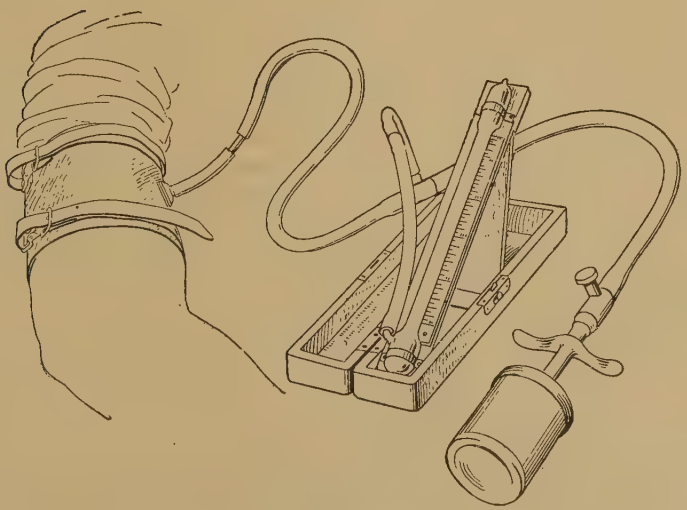

FIG. 283.—Oliver mercurial compressed air manometer. (*After Norris.*)

obliteration of an artery in terms of a column of mercury, an aneroid pressure indicator or compressed air activating a fluid column, respectively.

An example of each is shown in the accompanying illustrations, (Figs. 281–283) which serve to typify the numerous blood pressure instruments in clinical use, all based upon the three principles mentioned.

In its clinical application,the brachial is the artery chosen for compression. This is accomplished by means of a standard 12 c.m. broad, flattened cuff placed upon the upper arm. To the cuff are attached two tubes: One connected with a small metal or rubber hand pump for the purpose of inflation, the other connected with the mercurial column or aneroid instrument. A great disadvantage of the latter is that the spring may lose its resiliency; as a result, the instrument becomes inaccurate. If standardized sufficiently often by comparison with a mercurial sphygmomanometer, the aneroid manometer is as useful for clinical purposes as the mercurial instrument. Its advantages are that it is made compactly, so as to be more readily carried about, and the dial can be read more conveniently than the millimeter markings in a column of mercury. In the compressed air manometer (Fig. 283), pressure from the hand bulb forces the liquid (mercury or colored fluid) from the bulbous reservoir into the manometer tube, where the pressure can be read off. The advantages are compactness and ease of operation; the chief disadvantage is lack of sensitiveness.

Arm cuffs are of two types: One stiff, made of leather; the other, soft and made of cloth or silk (Fig. 281). Though either can be used, I prefer the soft cuff because it is pliable, and can be more readily and quickly placed in position.

Blood pressure estimations of the brachial artery should be taken at the level of the heart. For this purpose, the elbow of the patient is placed comfortably upon a desk or table. The cuff is then snugly applied to the bare arm. The arm should be relaxed, otherwise muscular tonicity tends to produce an incorrectly high blood pressure reading. Edema also causes abnormally high blood pressure, because much of the pressure within the cuff is used up in displacing the œdema.

The introduction and use of the sphygmomanometer have shown that the estimation of blood pressure by radial palpation alone is erroneous and untrustworthy. There are various methods of determining the systolic blood pressure by manometric readings; these are (1) the palpatory, (2) the visual, (3) the graphic, (4) the auscultatory.

According to (1) the palpatory method, the systolic blood pressure is that degree of cuff pressure exerted on the brachial at which the radial pulse becomes no longer palpable. (2) The visual method consists in compressing the brachial beyond the point of obliteration, and then gradually lowering the pressure by cuff deflation until the first mercurial oscillation becomes visible; this point marks the systolic blood pressure. In the (3) graphic method, the mercurial manometer has an additional connection with a rubber

bulb inclosed in a hermetically sealed small glass globe; the latter is attached to a recording tambour, so that differences in the mercurial oscillations are transmitted to the tambour and recorded on a revolving drum.

4. The auscultatory method (Fig. 281), first used by Korotkow, is the one usually employed because of its accuracy. The cuff is applied and inflated until the brachial is obliterated, that is, beyond the point at which mercurial oscillations are visible. The stethoscope is then placed over the brachial at the bend of the elbow, and the cuff is gradually deflated until sharp distinct taps are heard; this marks the systolic blood pressure. In typical instances, it is possible to separate the auscultatory phenomena into five distinct "phases." The first is the one just described and, as noted, establishes the systolic pressure. The artery beneath the cuff being empty, the first pulse wave produces sudden arterial tension with resultant short, popping sounds. With a gradual drop in blood pressure by further deflation, the sharp tap gives way to a murmur, somewhat resembling a superficial, rough pericardial friction sound. This marks the second phase. It is probably caused by sufficient blood passing under the cuff to produce swirling currents which mask the click of the first phase. By still further deflation and consequent lessened pressure on the brachial, this murmur ceases and the third phase begins. It is marked at its beginning by short tapping sounds; with continued deflation of the cuff, these gradually change to muffled taps. This is usually regarded as the beginning of the fourth phase. It seems probable that, with the gradual approach of diastolic pressure, a steady arterial stream begins to flow beneath the cuff between the arterial pulsations; this acts as a kind of buffer and thus produces the muffled, dull sounds characteristic of the fourth phase. The dull note is finally extinguished by a constantly increasing column of blood in the artery, until all sounds cease. This marks the fifth or last auscultatory phase. The usual difference between the fourth and fifth phases is from 5 to 8 m.m. of mercury.

The various phases as outlined are not always well defined nor distinguishable. As measured in the mercurial column, the average millimeters of pressure produced by the various phases are 14, 20, 5, 6, from the first to the fifth phases, respectively (Goodman and Howell). There are several variations from the normal. If the brachial artery is small, the separate phases may be indistinct and inaudible. Again, the first phase may not be represented by a sharp click, but by a murmur. Occasionally, the second phase is entirely absent, especially in aortic disease with high systolic blood pressure. Such differences probably depend upon the varying strength of arterial eddies interfering with the usual sharp taps of sudden arterial distention.

The maximal normal blood pressure is still a matter of dispute. I regard a systolic blood pressure between 115 and 135 m.m. of mercury in an adult as within normal limits. In a statistical table by Fisher representing 19,339 accepted candidates for life insurance, including ages from 15

to 60, the average systolic pressure was 129 m.m. of mercury. There is a gradual rise in systolic and diastolic blood pressure with advancing age; this probably depends upon associated vascular pathological changes in the cardio-renal-vascular system.

There are certain physiological variations which must be born in mind when making blood pressure estimations. The systolic blood pressure is increased by expiration; in forced expiration this may amount to 5 to 10 m.m. There is also a slight diurnal variation accounted for by differences in physical and psychical states of the individual at various times of the day. The blood pressure is lowest during the first part of natural sleep. The ingestion of meals has a varying influence upon blood pressure; this depends upon different degrees of vasodilation of the abdominal vessels with compensatory superficial vasoconstriction.

There are certain factors which require brief comment because of their influence upon blood pressure. Moderate amounts of alcohol do not regularly raise the blood pressure in man. In the experimental animal, large doses produce a fall in blood pressure, due to a depressant effect upon the vasoconstrictors and upon the heart. During smoking, there is a temporary rise of blood pressure from stimulation of the peripheral and central vasoconstrictor mechanism. Excitement—pleasurable or otherwise—sometimes produces a temporary rise of blood pressure. Exercise and muscular exertion have a similar effect; in young healthy individuals the blood pressure soon drops to its usual level; in the middle aged and old, the rise of blood pressure represents a more marked reaction and lasts longer. Cyanosis may in itself be a cause of abnormally high blood pressure; hence therapeutic measures, like venesection and digitalis (which tend to diminish cyanosis) may directly decrease existing hypertension. This is an important consideration in the treatment of decompensation and hypertension in which cyanosis forms a conspicuous element. Another factor in the production of hypertension in some cases of arterial disease is that due to hypertonus; this possible cause for error, however, can be controlled and obviated by repeated compression of the artery with the cuff.

In general, frequent blood pressure estimations are necessary in order to arrive at the correct blood pressure of the individual, and to obviate some of the disturbing physiological and other factors that have been mentioned.

I shall not enter into the still much debated question of the basic cause of clinical hypertension. That it is fundamentally due to constriction and spasm of the arterioles—the vascular peripheral bed—seems highly probable. There are, however, many factors which undoubtedly cause vaso-constriction of the arterioles. Some of these are psychic,—for example, emotional disturbances and mental strain. Some are probably of endocrine origin, for example, the hypertension at the menopause. Others again are organic; this applies especially to chronic glomerular disease of the kidneys and to aortitis with associated general vascular disease. While the statement may

be true that vascular disease can produce hypertension, and hypertension, vascular disease, in the majority of instances we possess no knowledge as to which is the primary factor nor do we definitely know how arteriolar disease acts to bring about hypertension.

For the purpose of studying abnormal blood pressure in organic cardio-vascular disease, I have found the classification below, embracing the vast majority of cases, of clinical value. Despite occasional overlapping, the prominent lesion or type is usually readily recognized.

I. { (*a*) Hypertensive cardio-vascular disease with myocarditis.
(*b*) Hypertension and myocardial insufficiency with labile vaso-motor mechanism.
(*c*) Uremia.

II. Myocardial disease and insufficiency without hypertension.

III. Valvular disease and myocardial insufficiency with and without hypertension.

IV. Senile and premature arterio-sclerosis.

I. (*a*) Hypertensive cardio-vascular disease with myocarditis includes some of the fairly well-defined groups of the older writers; for example, Huchard's presclerosis, Gull and Sutton's arteriocapillary fibrosis, von Basch's angio-sclerosis. It is becoming increasingly evident that the pathological basis of hypertensive cardio-vascular disease lies chiefly in disease affecting the arterioles, especially of the heart, kidneys and brain. As concomitant changes in the heart, there may be cardiac hypertrophy (mainly of the left ventricle), and moderate or advanced aortitis. The systolic blood pressure commonly varies from 180 to 230 m.m. of mercury; the average, 190 to 200. It is important to note that there may exist no parallelism between the degree of hypertension and the extent of kidney involvement, and that hypertension alone need not necessarily be of symptomatic or prognostic importance. The average diastolic pressure of this group is about 100 m.m.; if renal involvement predominates, diastolic pressures of 120 or over are found.

I. (*b*) **Hypertension and Myocardial Insufficiency with Labile Vaso-motor Mechanism.**—This group consists principally of patients past middle life with only moderate hypertension and with normal or moderately elevated diastolic pressures. The chief characteristic of the blood pressure is its marked daily variation, which may be as much as 30 m.m. of mercury. The symptoms referable to myocardial insufficiency are mild. The patients are usually stout or obese men; the prominent physical signs in the chest are those of bronchitis and emphysema. The palpable arteries are not thickened, and nephritic symptoms not marked despite the presence of a slight amount of albumin and a few casts in the urine. Pretibial edema is absent or only very slight.

I. (*c*) **Uremia.**—The cardinal signs and symptoms in patients in this group are headache, nausea, vomiting, varying grades of pallor, attacks of paroxysmal dyspnœa, precordial distress and nocturnal polyuria. There are retinal changes of varying degrees. The phenolsulphonephthalein test

shows diminished output in two hours, the average being between 15 and 30 per cent. As a result of renal test meals containing weighed amounts of water, salt, carbohydrate and nitrogenous constituents (Chapter XXVI), and of chemical examination of the blood, we find as a rule low, fixed specific gravity for the day and night urines, decreased elimination of salt, water and urea in the urine, and abnormal amounts of non-protein nitrogen and of uric acid and creatinin in the blood. When uremic dyspnœa is marked, there is evidence of diminished blood alkalinity (so-called acidosis). This may be roughly estimated by determining the amount of bicarbonate of soda, administered internally, required to render the urine alkaline. Other more refined and direct methods consist in determining the carbon dioxide content of the alveolar air by the Priestly-Haldane bag or by testing the blood chemically according to the Van Slyke method. The differentation between the "cardiac" and "cerebral" types of hypertension can sometimes be made by careful observation of the diastolic pressure, which presumably serves as the better index of the peripheral resistance. The diastolic pressure is between 120 and 140 in the "cerebral" cases, *i.e.*, in those who suffer from such typical uremic signs as headache, vomiting and retinal changes; it is considerably less in those in whom "cardiac" manifestations are especially prominent.

**II. Myocardial Disease and Insufficiency without Hypertension.**—Patients in whom myocarditis is the predominant pathological condition show little or no hypertension. The diastolic pressure is sometimes quite low, so that, even with a normal systolic, there is an increase in the pulse pressure and hence in the cardiac load.

**III. Valvular Disease and Myocardial Insufficiency with and without Hypertension.**—The valvular lesions, that is, those without evidence of general arterial disease in which the systolic blood pressure is high, are rheumatic affections of the aorta, especially aortic regurgitation. In these, the blood pressure may be between 180 and 200 m.m.; the diastolic pressure is abnormally low, the average being 40 to 25; it is occasionally zero. There thus exists a very marked cardiac overload. It has been shown that in this valvular lesion, the systolic blood pressure in the popliteal is often much higher than in the brachial. This difference I have found to be as high as 50 m.m. of mercury in individual instances. The normal difference between leg and arm blood pressure is from 5 to 10 m.m.; hence the diagnostic importance of measuring the arm and leg blood pressures in cases of suspected aortic regurgitation in whom such usual clinical signs as typical murmurs and the Corrigan pulse are absent.

Unless arterial disease or cyanosis is present, mitral lesions are unaccompanied by hypertension. With beginning heart failure, the systolic blood pressure may become subnormal. In combined valvular disease of the mitral and aortic valves, blood pressure depends upon the clinically predominant lesion. For example, if aortic regurgitation be the more prominent, the

blood pressure will be characteristic of that lesion; if the mitral predominates the pressure will be normal.

**IV. Senile and Premature Arterio-sclerosis.**—Under this caption are grouped those cases with tortuous and thickened visible arteries. The heart valves and aorta present various degrees of intimal thickening and lime deposits, the main coronaries and their branches are thickened, there is marked myo-fibrosis, the heart may be small or only moderately enlarged. Such patients often have slight hypertension or even normal blood pressure, unless renal involvement is a marked clinical feature.

In addition to the foregoing classified groups of organic cardio-vascular disease, several other important diseases and conditions which have a direct bearing upon blood pressure estimations require brief description.

**Exophthalmic Goiter.**—A characteristic of this disease is the variability of the blood pressure readings; there are marked differences from day to day. When the disease is of moderate severity, hypertension is the rule, the range being between 160 and 180 m.m. of mercury. Extreme hypertension without accompanying cardio-vascular or renal disease is rare.

**Lead Poisoning.**—During attacks of lead colic there is usually a rise of the systolic blood pressure. This is attributable partly to pain present during the attack, but chiefly to spasm of the peripheral arterioles. Plumbism which causes disease of the kidneys and arteries, and ends in nephritis and arterio-sclerosis naturally produces hypertension because of the presence of these lesions.

**Increased Intracranial Tension.**—Hemorrhage, meningitis and brain tumors are the usual causes of this condition. As a consequence of increased intracranial tension, cerebral anemia results, and with it, an increase—sometimes marked—of the systolic blood pressure. In addition to the hypertension, other manifestations of this cerebral anemia may consist of headache, vomiting, vertigo, choked disk and true bradycardia. Apoplexy is usually accompanied by hypertension; hypertension, if already present, is sometimes increased. When the blood pressure continues to rise after a stroke, it is usually evidence of increased intracranial pressure from continued bleeding.

**Cyanosis** is so frequently found in broken compensation that its effect in producing hypertension requires special consideration. Its action may be ascribed to an effect, in a milder degree, similar to that of the blood in asphyxia. In the latter, direct stimulation of the vasomotor center is assumed. Cyanosis in itself may frequently account for hypertension in decompensation from any cause, for, with decrease of cyanosis, the blood pressure often returns to the normal and remains so. Therefore, by treating cyanosis, diverse therapeutic measures such as digitalis medication and venesection may have a direct and beneficial effect upon hypertension.

**Functional Hypertension, Functional Hyperpiesis, Essential Hypertension.**—Besides hypertension due to known pathological arteriolar and

vascular changes, there remains a group of patients in whom at present no pathological basis for hypertension can be found. This condition has been termed functional hyperpiesis, or functional hypertension. In this group may be mentioned females approaching the menopause and a few sufferers from gastric disturbances of a neurotic nature. The following is a typical case:

Female, age 55, menopause 8 years ago. Some years previously she had gastric symptoms—belching, dizziness and hunger pains. Her present complaints have lasted several months; these are dizziness, slight nausea and at times belching. Dyspnœa, edema and urinary changes are absent; the sodium chloride, water, urea excretion and phthalein output are normal; the Wassermann blood reaction is negative. The amount of non-protein nitrogen in the blood is also normal. Physical examination reveals no evidence of cardio-vascular disease. Despite all these findings the systolic blood pressure is regularly about 200; the diastolic, normal. It is possible that such cases are due to extreme susceptibility of the vasoconstrictor center to reflex influences. Until more of these cases have been studied by means of chemical examination of the blood and by functional urinary tests, and have been followed to necropsy, for the present they must be provisionally regarded as of functional, non-organic origin.

**Hypertension at the Menopause.**—In the entire absence of any physical or organic causes for hypertension, these patients resemble the functional hyperpiesis group just described. They are apt to suffer from the usual sweats and flushes incidental to the climacteric. Apparently vasomotor instability is an important factor in the hypertension. With increased knowledge, the fundamental etiological factor may be found in some disturbance of the endocrine glands.

**Functional Hypotension.**—This term applies to a group of adults presenting no evidence of organic disease and in whom the systolic blood pressure is about 100 in males, 90 in females. Contrary to what is generally believed, some of these individuals are of robust physique. They never suffer from decompensation. I have not found gastroptosis or constipation significantly frequent in them: They often run a symptomless course and are only accidentally discovered in the course of routine clinical examinations. If symptoms be present, they consist of rapid fatigue following moderate exertion; the vasomotor system is unstable and susceptible to nervous influences of various kinds. The patients flush or become pale readily. They are excitable, often become dizzy or complain of feeling faint; indeed, their symptoms overlap to a great extent those described under "Weak Heart" (Chapter XVIII).

## BLOOD PRESSURE IN CARDIAC ARRHYTHMIAS

It is sometimes important to estimate blood pressure when various types of pulse irregularities are present. The method to be employed in the commoner of these requires brief description.

**Sinus Arrhythmia and Heart Block.**—Since the beats at the wrist are of equal force, only the routine methods are required for the blood pressure determination.

**Alternation.**—There is a rhythmic sequence of stronger with weaker beats. The systolic blood pressure of the stronger contractions can be estimated in the usual manner. The diastolic pressure can also be estimated in this way if it be higher than the systolic of the smaller beats. This fact is readily determined during the course of the examination by palpating the radial; the number of radial beats which come through will then be just half those at the cardiac apex.

**Coupled Rhythm.**—The tension of the initial beats of the couplets in auricular fibrillation with coupled rhythm is not identical, but the difference is usually slight, so that for our purpose the coupling of auricular fibrillation may be grouped with that occurring in extrasystoles. The systolic blood pressure of the initial stronger beats is calculated in the usual fashion. If the diastolic pressure of these be greater than the systolic pressure of the weaker contractions of the couplet, then again the usual routine in measuring the diastolic pressure of the stronger is followed.

When extrasystoles occur frequently and at irregular intervals, it is necessary to use a special method similar to that for auricular fibrillation (see below) in order to estimate the systolic blood pressure.

In auricular fibrillation with gross irregularity in the force of the beats, the ordinary routine cannot be applied, for it can only estimate the systolic blood pressure of the strongest, and the diastolic of the weakest beat at the moment that the blood pressure is taken. When all ventricular contractions are propagated as pulse waves, and the radial beats on palpation feel approximately equal in force, my observations have shown that the pressure of the individual beats do not usually vary more than 10 m.m. of mercury; hence the ordinary method of estimating the systolic levels are sufficiently accurate for clinical purposes. With extreme irregularity in the strength of the radial, and with many frustrane and abortive beats, various methods have been devised to arrive at an approximation of the actual blood pressure. According to one method, that of James and Hart, the cuff and stethoscope are first applied in the usual manner. These observers have found that, although the rate is irregular the number of ventricular contractions for each minute is approximately the same. Their procedure is as follows: The number of radial beats which pass under the cuff between pressures of 150 and 140 m.m. of mercury, for example, are counted, as well as the corresponding number of ventricular contractions occurring during the same time. The difference between these represents the number of frustrane or abortive beats (pulse deficit). Similar observations are then made for systolic blood pressures between 140 and 130, 130 and 120, etc., until the systolic blood pressure of all the beats which pass under the cuff have been taken. The number of palpable radial beats found at the various blood pressures is multiplied by the

highest systolic limit set for that group; these products are added and then divided by the corresponding heart rates at the apex. The result gives the "average" systolic blood pressure. One objection to the method is that the "average" includes many abortive beats (called by the authors pulse deficit) which have no effect upon the circulation since they propel no blood through the arteries. Another plan is to average the effective beats only, *i.e.*, those that actually have some effect upon the circulation by the production of pulse waves. This is attempted in the "fractional" method of Kilgore, according to which the diastolic pressures are also calculated. The systolic and diastolic pressures are plotted on a chart, the number of beats occurring at the various systolic and diastolic pressures being marked by points; these are then connected so as to form a smooth graph. In this manner it is possible to make fairly exact estimations of the blood pressure in auricular fibrillation.

**General Remarks on Hypertension.**—Reference is here made to the hypertension itself and not to accompanying cardio-vascular, uremic or other symptoms.

Mental calm, avoidance of excitement, diversion are the first and perhaps the most useful therapeutic agents in hypertension. It requires the skill and phychological insight of the physician to understand best how to bring this about. The physician must naturally individualize. Some people for instance actually have added nervous symptoms if told they must take a prolonged rest and be absolutely quiet. Some exercise and perhaps games like golf may be allowed in appropriate cases. The livelihood of others depends upon their presence at business at least part of the day or several days a week. To these, week ends in the country or leaving business early daily may answer the purpose. To hard working people, perhaps lighter work can be prescribed. It is never wise to let neurotic individuals know how high their blood pressure is. It simply adds to their worry, subserves no useful purpose and tends to bring patients to unskilled hands who claim most wonderful panaceas for hypertension but who possess no medical knowledge.

## THERAPEUTICS OF HYPERTENSION

Before remedial measures are considered, it is well to emphasize that hypertension is often a conservative or compensatory process. As a corollary, it follows that hypertension itself does not necessarily require medication, for it is usually but an index of the underlying cardio-vascular or cardio-renal mischief, or of some functional or endocrine disturbance. It is the basic causative factor which requires therapeutic attack. Another important consideration is that remedies acting upon the normal individual (or animal) may have an entirely different effect in organic hypertensive disease. This, I believe, is the main reason for disappointments in the attempt to reduce blood pressure by vasodilators. They have been used innumerable times to decrease hypertension, in most cases with no or but slight temporary results.

Exceptions will be noted later. Their inefficacy may probably be ascribed to the type of the pathological change underlying hypertension. For example arterioles considerably thickened by disease or in a state of hypertonus can scarcely be influenced by drugs that exert their dilating power upon pliable arteries or under normal conditions of nerve tone.

In some hypertensive cases, there occasionally occurs a temporary rise of blood pressure beyond the usual level for these individuals. This rise is sometimes marked by dyspnœa, precordial pain, headache and vomiting. It is in these that relief from abnormal hypertension is especially desirable.

The drugs usually employed in the reduction of blood pressure are nitrate of soda, amyl nitrite, nitroglycerin, benzyl benzoate, spiritus ætheris nitrosi, erythrol tetranitrate and mannitol. In urgent cases, amyl nitrite in 5 minim pearls may be administered. Regarding nitroglycerin, the doses usually prescribed, from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain three times daily, rarely result even in temporary blood pressure reductions. If its administration is not followed by dizziness, I am in the habit of prescribing much larger doses, as much as $\frac{1}{25}$ of a grain three times a day. In an experimental series of cases, I gave as much as $\frac{1}{10}$ of a grain hypodermically. In those with uremia, the effects upon the blood pressure were disappointing. Even these large hypodermic doses had only an occasional or fleeting effect upon the blood pressure or symptoms. In others of this series, no constant result following nitroglycerin could be determined. Sodium nitrite, when efficacious, seems to have a more lasting effect. Erythrol tetranitrate has also been used with indifferent results. I have observed the best results when the vasodilators were employed in those individuals whom I have grouped as "hypertension and myocardial insufficiency with labile vasomotor mechanism" (q. v.). These patients are rarely uremic; the hypertension is relieved not only temporarily but sometimes for a prolonged period.

The action of the iodide of potash, a drug frequently prescribed for the relief of hypertension, is still in dispute. In view of our knowledge that syphilis is a frequent cause of cardio-vascular disease, the occasional good effects of the iodides are to be ascribed only to its action upon the underlying luetic disease.

Venesection in all cases of hypertension with urgent symptoms is an excellent temporary therapeutic measure, especially in plethoric and cyanotic patients. From 400 to 800 c.c. of blood should be withdrawn.

Hydrotherapy, hot packs, oxygen, carbon dioxide, or hot baths, or the electric light bath, has been advocated to reduce hypertension. In so far as they promote diuresis and perspiration, and thus rid the body of toxic material, warm packs and baths in those not uremic may have some direct therapeutic value. The incidental effect upon the vasomotor system may also be of benefit. Oxygen and carbon dioxide (Nauheim) baths are followed by varying and inconstant effects upon the blood pressure (Chapter XX). The indications for these gas-impregnated baths in hypertension depend

upon the degree of decompensation, and in the main, follow those indications already discussed for that condition. If decompensation is extreme, baths are contraindicated; if decompensation is mild, and observation from one bath shows good effects upon the blood pressure or symptoms, they may be continued. Even in those in whom there has been a reduction of blood pressure in or immediately following the baths, the reduction is slight and transient, so that the beneficial effect is probably to be attributed to an action upon the general circulation rather than upon the blood pressure.

The effect of electric light baths, when efficacious in the reduction of hypertension, may be ascribed to the heat, to favorable vasomotor change, to incidental rest and to enforced quiet which form part of the treatment.

**Diathermy.**—A special application of the D'Arsonval current to the precordium is another method employed as a therapeutic agent in hypertension. Some of the reports of its effect are exceedingly enthusiastic; claims are made, for example, that hypertension is permanently relieved. In some cases which I have observed in whom diathermy was used by other practitioners, the blood pressure remained at its usual level; there was no change whatever in the symptoms.

Percussion over the seventh cervical vertebra has been used, the object being to exert a reflex influence upon the vasodilators and thus upon the blood pressure. The effect of this procedure is, to say the least, exceedingly problematical and hypothetical.

Absence from business, and treatments at Spas have undoubted marked therapeutic value in many cases of hypertension. The reasons are obvious. Patients are away from their ordinary environment; their routine of life approaches a more normal and physiological standard; excitement and nervous tension become almost negligible factors.

The amount of exercise that should be allowed depends chiefly upon the cardio-vascular disease and upon the symptoms, rather than upon the degree of hypertension. At no time, however, should violent or fatiguing exercise be permitted. The amount of rest is also based upon the same criteria. For example, an otherwise active business man still capable of some physical exertion, who frets and becomes irritable when absolute rest is enjoined, may be permitted to attend to business for a short period each day. The week's work should be broken by one or two days of rest; or if the patient's condition allows it, by mild exercise out of doors, especially walking. Exercises of any type should be controlled by the state and degree of cardiac compensation, and by careful observation of the effect of exercise upon the individual.

Hypotension in itself rarely requires medication. When combined with the symptoms of vaso-motor instability, appropriate therapy should be administered (Chapter XVIII). Accompanying neurotic symptoms, when present, require appropriate medication. The extract of suprarenal gland may be used as a temporary expedient to raise the blood pressure.

**Venous Blood Pressure.**—Various instruments for this determination have been devised. Glass chambers, cemented to the skin (Hooker), or small springs and pelottes (Oliver, Sewall) appropriately connected with manometers are placed or connected over a prominent vein in the arm. The pressure is then read from the manometer scale. Another method (Moritz and Tabora) consists in plunging a needle connected with a manometer scale directly into the vein at the bend of the elbow. An approximate clinical method of determining venous pressure is that of Gaertner. The relaxed or flexed left arm of the patient is slowly raised until the veins at the bend of the elbow collapse. The height of elevation above the level of the right auricle (the upper border of the left fifth costal cartilage at the sternal junction, according to Gaertner), measures the venous pressure in millimeters of blood, approximately that of water. According to the v. Recklinghausen method, one hand of the recumbent patient rests at the side of the bed, the other on his thigh. If the veins of both hands are empty the venous pressure is low; if full, it is high: If the veins of the hand on the thigh are empty and those of the hand on the bed are full, venous pressure is normal.

N. W. Brown has devised a simple method by which the usual mercurial manometer may be used. Water or a somewhat heavier fluid like bromoform (sp. gr. 2.5) is used instead of mercury. The vein employed, usually on the back of the hand must be at the level of the heart. The tube is connected with a small saddle-shaped cup 2 c.m. in diameter covered with very thin rubber tissue, and loosely applied so as to allow free oscillation without tension. Manometer pressure is regulated with a thumb screw. Pressure in the manometer is raised several centimeters and the cup or capsule is placed upon the vein with sufficient pressure to obstruct the return flow. The pressure in the manometer is allowed to fall by opening the needle valve. When the venous pressure equals the manometer pressure, the vein above the capsule will rapidly fill, a reading at this time will give the approximate venous pressure (N. W. Brown).

Although the subject of venous pressure has not been exhaustively studied, it would appear that a venous pressure of 10 m.m. or less of water is within normal limits. Higher venous pressures are found in decompensated cardiac lesions, and roughly correspond to the degree of decompensation; a pressure from 10 to 20 m.m. suggests moderate cardiac failure; from 20 to 30 or over, indicates severe cardiac failure and venous stasis.

**Capillary Circulation.**—For many years, abortive attempts have been made to study the capillary flow and the capillaries themselves in human beings. A simple method has recently been devised by which this can be satisfactorily accomplished. The technique consists in placing the junction of the cuticle and nail covered with a drop or two of transparent oil on the stage of the microscope under the objective. A 50 or 100 Watt electric bulb is so focussed as to strike the finger end at an angle of about 45 degrees. The objective is a 16 m.m. lens, the oculars, a number 5 or 10; the capillaries

are thus magnified 50 to 100 fold. The skin capillaries form straight or tortuous loops.

The method is still too new to permit definite clinical deductions. Some interesting observations regarding capillary stasis in shock and in cardiac decompensation, the capillary flow in vasomotor instability and in hypertension have already been made. They require further confirmation before their clinical value is fixed. Indeed, a recent observer (Boas) claims that there is no actual pulsation visible in the capillaries and that arterial pulsation ceases in the end arterioles.

In this chapter it has been shown how the circulation may be followed through the larger arteries, capillaries and veins. Each will probably come to have its special value in helping the clinician assess in what system—arterial, venous, or capillaries—lies the important defect in the blood flow.

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## CHAPTER XXVI

## THE HEART AND CIRCULATION IN SPECIAL DISEASES—THE HEART IN DIPHTHERIA—THE TOBACCO HEART—THE AVIATOR'S HEART—THE CIRCULATION IN SURGICAL SHOCK—SURGERY AND THE ARRHYTHMIC HEART, WITH AND WITHOUT DECOMPENSATION—SURGERY AND HYPERTENSION—THE "FIBROID" HEART

**The Heart in Diphtheria.**—The pathological changes occurring in the heart in deaths from diphtheria have already been described (Chapter IV).

In severe diphtheritic infections, marked destruction of the myocardium may result in death. In one instance, a girl of 10 with severe diphtheria and heart block, leucocytic infiltration in the junctional tissue and in the ventricular musculature was found. In another instance, a child developed heart block during the course of diphtheria. Electrocardiographic examination several years later still showed the arrhythmia; however, there were no cardiac symptoms. I have personally observed a child 8 years old with very toxic diphtheria, in whom, after a few days, there were attacks of convulsions coincident with slow rhythm. Polygraphic tracings showed regular radial and ventricular activity at the rate of 40 per minute; the venous tracing was unsatisfactory. Heart block was diagnosed. The child died one week later. Necropsy was not obtainable. From the virulent course of the disease, heart block seemed due to toxic degenerative myocarditis.

Milder toxic destruction of the myocardium in diphtheria is evidenced by the usual signs of myocardial insufficiency, especially by dyspnœa. Another evidence is the presence of arrhythmias—heart block, auricular and ventricular extrasystoles, auricular fibrillation, tachycardia. These types may frequently interchange. They are apparently indicative of myocardial degeneration. Pathologically, the muscle cells undergo fatty degeneration, the valves and endocardium are rarely involved. With reference to the arrhythmias, it must be remembered that, as in other acute febrile and infectious diseases, cardiac irregularities may occur at the onset or crises, without any evidence of cardiac degeneration (Chapters X, XI). Such irregularities at these times are probably the effect of toxins on the normal neurogenic cardiac control; they are harmless and rarely require medication. It is therefore important to differentiate the innocuous from the dangerous arrhythmias in diphtheria. With our present knowledge, this is the best accomplished by a careful and complete examination of the entire cardio-vascular apparatus, and not alone by a consideration of the types of arrhythmias. The

presence of cardiac failure, slight or severe—dyspnœa, cyanosis, edema, cardiac dilatation—indicates that any type of irregularity, except physiological sinus arrhythmia, is of serious or even ominous import.

The study of the heart in diphtheria is further complicated by the occasional occurrence of unexpected, sudden death. This is usually regarded as due to paralysis of the cardiac nerves. Some of these deaths can, I believe, be explained on the assumption of a destructive, progressive myocardial degeneration with slight or no symptoms during life, or with the cardiac symptoms masked by those due to the diphtheria itself. In other cases in which myocardial damage is not demonstrated at necropsy, these fatalities may possibly be regarded as anaphylactic phenomena affecting the cardio-inhibitory center or the intracardiac nerve supply.

Treatment for the diphtheria itself should be carried out along the well recognized lines of antitoxin injections. Where cardiac failure is present or imminent, vigorous circulatory remedies (Chapter XX) should be used, chiefly digitalis and the caffein derivatives. The presence of arrhythmias—even heart block—is no contraindication to this general rule. An attempt should be made to relieve the block by atropine sulphate, best given subcutaneously in full physiological doses.

**The Tobacco Heart.**—As already stated (Chapter IV), I regard the effect of tobacco poisoning upon the heart as being very probably the result of the selective action of nicotine upon the nerve mechanism of the heart, rather than upon the musculature and endocardium of the heart itself. In addition, evidence of organic cardio-vascular disease is lacking. The assumption of a neurotoxic poison satisfactorily accounts for the various arrhythmias—sino-auricular block, extrasystoles, auricular flutter and even auricular fibrillation—which can be produced by tabagism. It also accounts for the clinical fact that in almost all instances in which tobacco is withdrawn, the arrhythmias soon disappear.

Precordial pain is one of the common as well as distressing accompaniments of tabagism. It seems due to disturbance in the coronary circulation and to referred irritation from excitation of the sympathetic ganglia. The types of pain and their therapy have already been described (Chapter XXIV). The diagnosis, symptomatology and therapy of cases of "tobacco heart" are given in Cardio-vascular Clinics. Brief allusion should again be made to the fact that during actual smoking, there is often a slight moderate rise of blood pressure, probably the result of stimulation of the peripheral and central vaso-constrictor mechanism.

**The Aviator's Heart.**—During the Great War, many studies were made on the effect of aviation on the heart and circulation. These observations will undoubtedly have great value in peace times, as aviation becomes more general.

Observations were at first disjointed because they were confined to cursory examinations during flight, and there was no method of checking up

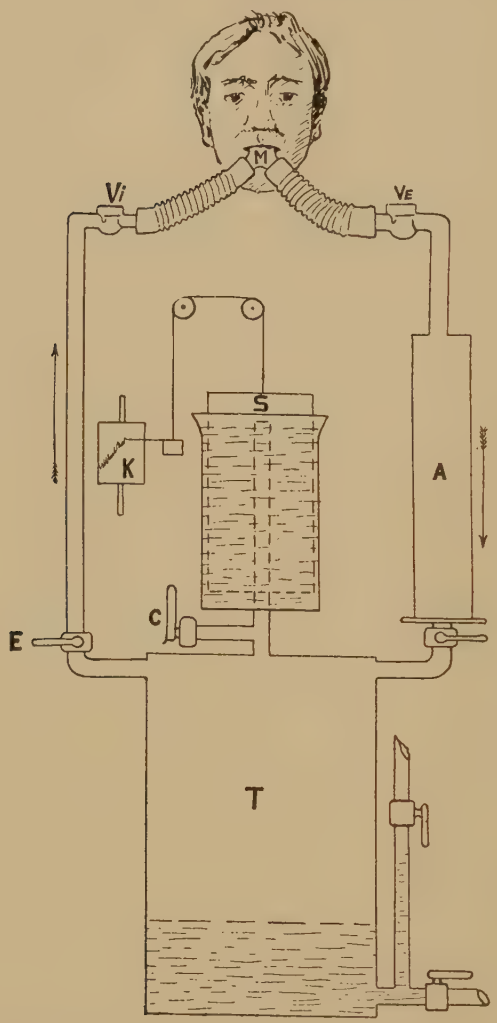


FIG. 284.—Diagram of the Henderson re-breather—the rebreathing apparatus employed in all routine tests of the aviator's ability to withstand low oxygen. It consists of a tank, *T*, of about 120 liters capacity. The volume of air is determined by the amount of water that is run into it. The man under examination continually rebreathes the air of the tank (a clip is placed on his nose) through the inspiratory and expiratory valves, *Vi* and *Ve*. The oxygen is thus consumed and reduced. The exhaled carbon dioxide is taken up by sodium hydroxid in the absorber *A*. The movements of respiration are recorded by the spirometer, *S*, connected to a smoked drum, *K*. As the oxygen is consumed and the air volume is thus reduced, the spirometer falls and the graphic record on the smoked drum rises. At the end of the test a sample of air is drawn from the tank and analyzed as a confirmation of the oxygen consumption and of the oxygen percentages (that is, altitudes) indicated by the graphic record. (*From Medical Studies in Aviation, Journal American Medical Association*, 1918, LXXI, 1382–1400.)

by refined methods such necessarily hasty examinations. It was soon discovered, however, that the atmospheric conditions of actual flying could be imitated in the laboratory, for oxygen deficiency in the higher altitudes was found to be the underlying cause of all the circulatory disturbances. Tests of the candidate's ability to fly were therefore instituted. This was at first accomplished by the so-called re-breathing apparatus (Fig. 284). Later, a large steel chamber with an air pump was devised, so that several candidates could be examined under any desired reduction of air pressure.

As was to be expected, it was found that individuals react differently to different amounts of oxygen deprivation in rebreathing. In some respects and up to a certain degree of "altitude" in the re-breather, there was found to be a physiological compensation for the lack of oxygen supply; this showed itself in the type of breathing and in changes of pulse rate and blood pressure. Most subjects breathed not only more frequently, but also more deeply—reactions which began quite early in some of the oxygen deprivation experiments, and which naturally resulted in increased pulmonary ventilation.

The physiological circulatory reaction in the oxygen-deprivation, rebreathing experiments resulted in increased cardiac and pulse rates, that is, in increased oxygen supply. A normal reaction constituted an increase of from 15 to 40 pulse beats per minute when the oxygen had been lowered slowly to between 7.5 and 6.5 per cent. Absence of such response denoted "either inability to react to a low oxygen of high altitudes and early failure, or that sufficient compensation is secured by increased breathing or blood concentration or both" (Schneider). Failure to respond is usually associated with poor tolerance for high altitudes. Occasionally, there is an abnormally increased heart rate; such reactions usually disqualify subjects for higher altitudes, although they may safely fly at lower levels.

Regarding the effects of altitude on the systolic blood pressure, the best type of response seemed to be that in which the systolic pressure remains unchanged until the amount of oxygen has been considerably reduced, after which point it gradually rises. Similarly, the best response in diastolic pressure is that in which it remains at the same level, or is slightly increased throughout the test. Occasionally, in those exhibiting poor reaction, first the diastolic, and then the systolic pressure fell; this is then accompanied by a slow pulse rate and fainting attack.

Careful roentgenographic studies were also made regarding the size of the heart in rebreathing tests. These observations proved that roentgenographic plates taken during various phases of respiration resulted in marked differences in the cardiac outline, especially in the transverse diameter, differences which ultimately depended upon how much the diaphragm was raised or lowered during inspiration and expiration, respectively. I have long since emphasized similar observations in routine fluoroscopy (Chapter XII). Although the

observers arrived at no definite conclusion, there was no evidence that there was any regular change in the cardiac diameter or contour as the result of oxygen deficiency during rebreathing experiments.

There were other important experimental observations of the aviator—for example, his visual, auditory and psychological reactions. These however are beyond our field.

From the studies that have thus far been made, one may safely conclude that the important primary effect of aviation upon the circulatory apparatus is a vaso-motor one; that circulatory failure when it occurs is primarily of vaso-motor origin, and is only secondarily due to the heart; and that lack of oxygen is the fundamental cause of the vaso-motor instability. This assumption further explains why aviators should be in prime condition, and why, when "stale" as the result of overwork and insufficient nerve relaxation, or as the result of improper "training" conditions, the vaso-motor nerve balance is more readily upset; as a consequence abnormal reactions under flying conditions appear sooner or become exaggerated with corresponding danger to the flyer.

**The Circulation in Surgical Wound Shock.**—I refer here not to circulatory failure from hemorrhage—slight or severe—but to so-called surgical wound shock, a condition described as "functional disturbance of the circulation which follows severe trauma or surgical operation and which progresses into a severe condition, even death. Upon post mortem examination, no pathological changes can be found to account for it" (Wiggers). Experiences during the late war have given renewed impetus to the study of this important subject. It is now generally recognized that surgical shock is a complex phenomenon and that it may arise from the most diverse causes. The most varied theories have been advanced to account for surgical shock, from one emphasizing psychic disturbances such as fear and insomnia (noci impulses —Crile) to those which seek the fundamental causes in the veins and peripheral circulation. The more widely held theories will be briefly presented subsequently.

Regarding some of the surgical conditions that have an influence in the production of shock, it has long been known that extensive burns, rough handling of the intestines during abdominal operations, cerebral concussion, etc., are such types. On the other hand, extensive muscle, tendon and bone operations (except upon the cranium) are not as apt to be accompanied by the manifestations of shock.

The clinical picture found in shock is the same no matter what the cause. The pulse is soft, rapid and often irregular. When shock is severe, the pulse may be slow and readily compressible. The systolic blood pressure is low, with a very small pulse pressure; the respirations are irregular and gasping; the patient is usually apathetic. These signs are quite similar to those found in massive hemorrhages, hence the extreme importance of correctly recognizing the etiological factor in a given case. Among other observations,

systolic blood pressure studies have been made, especially for the purpose of determining any clinical correlation between blood pressure readings and the onset or threatened onset of shock. The general statement may be made that when blood pressure has fallen from the normal to about 100 m.m. of mercury, it is evidence of the imminence of shock, especially if accompanied by a low diastolic or small pulse pressure. It should be emphasized, however, that low blood pressure itself is but a part of the clinical picture, and that other manifestations such as rate of heart action, cold extremities, presence of cyanosis or pallor, or irregular breathing must be considered in order to determine not only the presence of shock but to roughly estimate its degree. Depending also upon the urgency and type of operation, the surgeon must decide upon the appropriate remedies as well as upon the advisability of further operative procedures.

As the result of physiological and surgical experimentation, and from clinical observations the following theories regarding the causes of circulatory failure in shock have been envolved.

1. **Shock from loss of circulating fluid—acute oligemia (Henderson)** occurs not only in hemorrhage with loss of blood, but also in such conditions as serous diarrhœas and abdominal parecentesis. This type of shock was at first attributed to fatigue and failure of the vaso-motor center; later experiments, however, have shown that that center remains intact and can be reflexly stimulated until an extreme degree of circulatory failure sets in (Porter). Shock in oligemia may perhaps be directly attributable to the toxic effect of the deprivation of large amounts of fluid.

2. **Abnormal Production and Absorption of Poisonous Proteids.**—This theory assumes that because of venous and capillary stasis found in shock, split proteid products are formed and act as toxic agents. Objections to this theory are that one does not know whether these by-products may not be the "normal" results, so to speak, of such stasis; besides, absorption is practically at a standstill during shock.

3. **Fat embolism** has been given as one cause of shock. It has been shown experimentally that when oil is injected intravenously and causes extensive embolism in the lungs, death immediately follows from plugging of the bronchi; when less extensive, there is a primary fall of arterial pressure which later rises (Wiggers). How often such experimental conditions are reproduced in the human being in the assumption of fat emboli as the cause of shock and death must depend upon clinical findings and autopsy studies. It does not appear likely, however, that fat embolism is a very frequent cause of shock.

4. **Venous Stasis in the Venous Reservoirs of the Chest and Abdomen.** The view that during shock "the patient bleeds into his own veins" is based largely upon the experimental observations that shock induced in animals by manipulation of the intestines is accompanied by marked turgesence and stagnation of blood in the mesenteric veins and intestinal walls. Such

venous stasis, however, was not seen by surgeons in the frequent abdominal operations in shocked soldiers during the recent war (Cannon).

**5. Anoci-association.**—According to Crile's theory of nerve exhaustion, there is impairment of the vaso-motor mechanism and exhaustion of the brain cells, suprarenal gland and liver. Porter has demonstrated experimentally that the vaso-motor center does not lose tone in shock and that it is not primarily exhausted. Furthermore, regarding the histological changes in the brain cells found by Crile, these may well be the result, not the cause of the circulatory failure. Besides, other observers have not considered such changes in the brain cells as definitely pathological. With reference to the assumption of suprarenal exhaustion, it may be stated that there is "experimental testimony that painful stimuli and asphyxia increase both the secretion of epinephrin and the percentage of sugar in the blood" (Cannon). These data would seem to controvert Crile's theory in a large proportion of cases. It should be added that when circulatory failure in shock is extreme and long-continued, one will naturally find exhaustion of all glandular and nerve structures.

**6. "Acidosis" (Henderson)** has been found in experimentally shocked animals; it has also been found in the human being in shock (Cannon). In "acidosis" there is a low carbon dioxide content in the blood. It does not seem likely that this condition is primary: It is probably the result of more fundamental disturbances found in shock, especially decrease in the amount of circulating blood.

**7. Stasis in the Capillaries.**—This assumption is based partly upon the clinical observation that in shock there is concentration of the blood, and stagnation of the corpuscles in the capillaries as compared with the veins (Cannon). The assumption is further based on the exclusion of other factors. The arterial system being comparatively empty and the veins not always overfilled, it is assumed that the blood must necessarily stagnate in the capillaries. Physiological data seem to show that the capillary bed is sufficiently expansile to act as a reservoir for the blood lost to the circulation in shock and that concentrated blood has a tendency to enhance this capacity.

**8. Blood Viscosity.**—Cold increases the H-ions in the blood ("acidosis") in shock; it may therefore increase blood viscosity and thus have a certain influence in enhancing capillary stagnation.

The following conditions—tachycardia and dilated heart—are included here not because they constitute shock in the true sense, but because they can readily develop into a condition closely resembling the shocked state.

**Tachycardia,** especially the paroxysmal variety (Chapter XI) may have a decided influence in producing a clinical complex in every way resembling shock. In those cases that I observed, this was especially true when abdominal operations were performed upon women who feared operation and who had slight tachycardia before anesthesia was begun. The correlation

between tachycardia and the symptom complex of shock is described in conjunction with Cardio-vascular Clinics.

**Dilated Heart.**—This assumption, often loosely used by surgeons and physicians as the cause of shock, has been described in another connection (Chapter VI).

From this multiplicity of theories regarding shock it is evident that many causes operate to produce it. Long exposure on the battle field and a chilled body may be one etiological type, surgical procedure in a frightened woman another. Weighing all data, it seems probable that capillary stasis and a decrease in the amount of circulating fluid are two of the most important dynamic factors, and that abnormal nerve influences play an important role in initiating and bringing about the circulatory failure found in shock. It should be strongly emphasized that, no matter what the cause, the heart is not the organ primarily at fault. As shown experimentally, "the effective right auricular pressure is low, the ventricular filling is diminished, the ventricular relaxation incomplete, and the output per beat consequently small. The heart rate is rapid but the minute volume is diminished. The arterial blood pressure is low and the peripheral flow is slowed" (Wiggers).

**Treatment of Circulatory Failure in Wound Shock.**—The most effective measure is the application of warmth. This may be accomplished by the use of hot water bags or bottles (care being taken not to burn the patient); by rolling the patient in warmed blankets; or where available, by an improvised covered cage furnished with electric lights. The foot of the bed or table should be elevated. Some surgeons find bandaging of the extremities of added value. Blood transfusion has been frequently used but does not possess any special advantage. Because the fall of venous pressure and of peripheral resistance are the two out standing circulatory features of shock, epinephrin, adrenalin and pituitrin—powerful vaso-constrictors—are apt to have a strong although temporary effect in overcoming vasodilatation. Epinephrin may be given intravenously. In order to make up for loss of circulatory fluid, saline solutions by means of the rectal Murphy drip, or physiological salt solution given intramuscularly or intravenously are of value. The rectal drip or intramuscular injections are preferable; the fluid, although more slowly absorbed, is more apt to remain in the circulation. When injected intravenously, it passes through the kidneys very quickly and hence does not increase the volume of the circulating fluid for any length of time. Besides, some surgeons believe that by thus quickly throwing so much added fluid into the circulation, the heart becomes distended and dilated, and circulation even more embarrassed. In order to have fluid remain in the circulation, heavier solutions such as glucose and gum acacia mixtures have been tried, especially in hospitals in the war zone during the recent war. Such solutions, however, have not been found of more value than the older routine methods; some of the injections, indeed, were followed by serious results, even death. To combat tissue "acidosis," bicarbonate of soda

solutions may be given intravenously or by the Murphy drip. Drugs acting directly upon the heart such as digitalis and strophanthin possess but little if any value. Caffein sometimes helps temporarily. Strychnine and camphor are of questionable utility.

**Surgery and the Arrhythmic Heart. Surgery in Hypertensive Cases.**—It is of course axiomatic that surgery should be practiced only for definite indications when heart disease is present or is suspected, that the operation should be as brief as possible, and that surgical intervention even in extreme cardiac failure is justifiable if it offers the one chance for life.

Whenever possible, operations on those with heart disease should be done under local anesthesia. Where general anesthesia is indicated the best anesthetic is ether alone. A mixture of gas and ether may be used occasionally. Chloroform is to be avoided.

It may be stated as a general rule that patients with decompensation from any cause withstand operations, even slight ones, poorly if they are nervously upset by fear or dread of the operative procedure (Chapter XIV). In such instances, nervous influences undoubtedly play an important part in further deranging circulation, usually by producing tachycardia and by increasing dyspnœa. Bromides or morphine, or both are the remedies to be given to these individuals before operation. In addition, all dread and fear must be removed by the tactful, sympathetic physician or surgeon.

Functional arrhythmias (Chapter XI)—that is, those not produced by organic cardio-vascular disease, require no other care than that usually given to the heart and circulation during routine anesthesias and operation. This statement applies even to heart block if of functional origin. Thus, I observed a case of complete heart block with rapid ventricular activity (see Cardio-vascular Clinics) operated upon for tuberculous peritonitis; he stood both anesthesia and operation very well; polygraphic tracings that I took during the operation showed that it had no effect upon the block. An important exception to the rule disregarding functional arrhythmias is to be made in cases of functional tachycardia due to fright. Unless the patient is sufficiently calmed so that fear is dispelled, serious post-operative circulatory consequences may occur (Cardio-vascular Clinics).

Unless the surgical condition is assumed to be the cause of the cardio-vascular disease, as for example in local infections, decompensation calls for postponement of any but imperative operative procedures until attempts have been made to restore compensation. It naturally requires the close and balanced co-operation of physician and surgeon to decide how long one may wait for restoration of compensation in surgical conditions which, by waiting, may at any time become dangerous to life; acute appendicitis with fever and signs of peritonitis is a case in point. The presence of various types of arrhythmias does not of itself change the operative indication unless indeed the cardiac irregularity is considered special evidence of decompensation. For example one need not necessarily postulate that heart block be so

regarded, for if such a patient has but little dyspnœa, no bronchial rales, no cyanosis and slight pretibial edema at the time of operation, he may stand even a serious operation very well. So also patients with auricular fibrillation and mitral stenosis if otherwise in a fair state of compensation and with no extreme tachycardia usually tolerate operations well.

Decompensated patients who are cyanotic are poor operative risks. Patients with hypertensive cardio-renal disease with but slight dyspnœa are comparatively good operative risks.

Up to the present time electrocardiographic observations have not helped in distinguishing the poor from good operative risks in those with cardiac disease. Electrocardiograms give invaluable information regarding arrhythmias and occasionally of myocarditis; but as so frequently emphasized in various connections, this is but one part of a cardio-vascular examination; we require other data in order to determine whether a heart is or is not compensating (Chapter XIV), that is to say, whether the circulation is being carried on efficiently or not.

**The "Fibroid" Heart.**—Brief mention should be made of the assumed connection between the fibroid uterus and the "fibroid heart." It had for a time been believed that cardio-sclerosis was a frequent accompaniment of this type of uterine tumor, especially when extrasystoles were also present. Careful post-mortem studies of women with myomata have disproved this theory; cardio-sclerosis was not found more frequently than in those of similar age without myomata. Those with fibroid uteri stand operative interference as well as women with other comparable operative conditions. Arrhythmias when present and of functional origin should not interfere with the usual operative indications.

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## CHAPTER XXVII

## THE HEART IN PNEUMONIA—THERAPY FROM THE CIRCULATORY STANDPOINT

Because of the frequency with which cardiac drugs are employed in pneumonia, I wish to review my impressions gained in the drug treatment of many cases of that disease. I shall not touch upon the bacteriological or chemotherapeutical aspects of pneumonia. Statistical study of various epidemics or methods of treatment have likewise not been dwelt upon. Indeed, it is because I feel that none of the methods of medicinal treatment has been followed by any demonstrable correlation of cause and effect, of therapy and cure, that I have discarded statistics as bringing us no nearer to the therapeutic and circulatory problems of pneumonia. A study of these therapeutic methods, some of them directly antagonistic in theory and practice, arouses doubt as to their possible efficacy. Mere mention of some of the types of treatment, many long since discarded, may therefore not be amiss. Creosote, alcohol, no treatment until the crisis, no treatment for hyperpyrexia, cold sponges or even tubs for the fever, no stimulation, stimulation at the crisis only, stimulation to prevent the dangers of the crisis, camphor in oil, caffein, digitalis, adrenalin, venesection, fresh air, cold air—all these are or have been popular.

"Stimulation" has been the usual therapeutic keynote, for the danger was always correctly conceived as circulatory failure. I have used all of the recognized cardio-tonics—digitalis, strophanthus, caffein, adrenalin, as well as those whose effect upon the circulation may well be questioned—alcohol, strychnine, camphor. Camphor I have used in smaller, repeated, as well as in single large hypodermic injections, though experiments have shown that camphor in oil had no effect in delaying or preventing death in artificially induced pneumonia. I have employed the other drugs mentioned in all possible forms and combinations; in heroic, in small and moderate doses, subcutaneously, intravenously, and internally, before, during and after the crisis. With possible isolated exceptions, I have yet to see any of the effects upon the circulation which follows the use of some of these drugs in the experimental animal, or in cardiac failure from cardio-vascular disease.

I believe the chief reasons for disappointment in pneumonia therapy are that we are dealing with an infection of varying virulence, and involving varying amounts of lung tissue. Therefore I have attempted, for clinical and therapeutic purposes, to roughly group and to treat pneumonia according to degrees of toxicity and extent of lung involvement. I consider the crisis a separate problem for therapy.

1. **Toxic Cases.**—In the extremely toxic group, typically exemplified by sharp onset, early delirium, subsultus, dry tongue, no pain, rapid pulse and breathing, my experience has been that attempts at "stimulation," a term which we shall apply to drug therapy used to combat and treat circulatory failure, are almost always without efficacy. These patients apparently defy all drug therapy, and sometimes die before the area of pneumonic involvement becomes clinically recognizable. Death ensues from acute toxemia. Stimulation, even if heroic, has not in my hands delayed or prevented the fatal outcome in any visible manner. The reason is presumably that the drugs do not and cannot combat the *infective* factor. Experiments throw an interesting sidelight upon this problem. It has been found that hearts perfused with pneumonic blood lose their efficiency, which is later restored by the use of normal blood. Whether in the human being the changes in the cardiac musculature are, or later become myocarditis are questions into which we shall enter later.

It is usually believed that in toxic pneumonia there is marked vasomotor failure, though others, as the result of animal experimentation, have held otherwise. I have only very rarely seen any therapeutic result from single or repeated injections of adrenalin given subcutaneously or intravenously. A like negative result has almost regularly followed the use of strophanthin and other digitalis bodies when the pulse was regular. That digitalis does localize itself and affect the heart muscle in pneumonia as in purely cardiac disease has already been shown by the usual electrocardiographic evidence of digitalization—a negative T wave in head I and III (Chapter IX). Despite this change in the electrocardiogram, I have found in rhythmic cases no evidence of any help to the circulation from digitalis—cyanosis was not decreased, dangers from edema of the lungs not reduced.

As a favorable prognostic sign, Gibson has emphasized a certain parallelism between pulse rate and systolic blood pressure; but others as well as myself have found his so-called law of no significance. The removal of 500 to 700 c.c. of blood by venesection gives occasional temporary relief in toxic cases; the benefit seems attributable to ridding the body of toxic material rather than from any primary circulatory relief.

2. **Area of Pneumonic Involvement.**—With no evidence of severe toxemia, slowly progressing consolidation confined to one lobe is usually not dangerous during the acute febrile period. Such are probably the cases in which no treatment or any treatment has its measure of success. If, however, there is sudden massive involvement of one entire lobe and part of or all of another, the inflamed area in itself offers a problem for therapeusis, aside from the question of toxicity or crisis; for the pulmonary circulation and pulmonary ventilation may be sufficiently interfered with to produce cyanosis and beginning circulatory failure. Here venesection in plethoric individuals may be indicated for the relief of venous stasis; "stimulation" has a better chance for success, for the problem approaches more nearly a purely circulatory

one. Cardiac tonics should be given in large doses from the very outset to combat, if possible, ever impending circulatory failure.

3. **The crisis** apparently marks that point at which there is more or less complete sudden destruction of the pneumococci in the lung, while lysis marks their more gradual destruction. I distinguish clinically two types of crises—the febrile, and that of sudden pulmonary resolution. These are not necessarily simultaneous. A febrile crisis alone is not dangerous, but sudden resolution of a consolidated area has always been regarded, and rightly so, as the most critical and dangerous state of the disease. At such times there is apparently a rapid flooding of the blood and lymph channels with the liberated toxic, pneumonic products. Absorption probably occurs quite readily because of the general liquefying process and because the lymph and venous radicals are released from the pressure of the solidified lung. In other words, chemical and mechanical factors come into play at a time when the toxic products are greatest in amount. The heart muscle is thus bathed liberally and continually with pneumonic poisons. Interference with pulmonary circulation, caused by the onset of resolution is doubtless an added burden to the right heart. The main dangers arising during the crisis are general, from sudden systemic toxic invasion; and local, from edema of the lungs. The local danger, from the circulatory standpoint, depends upon the amount and rapidity of resolution, and the consequent pulmonary edema. An extremely important factor, scarcely mentioned in the literature, is the presence or absence of expectoration at the crisis. Pulmonary edema complicates and accompanies resolution more frequently if expectoration is absent; while if expectoration is profuse, there is increasing opportunity for pulmonary aeration, and less for edema. I insistently try to make the patients cough in the hope of loosening and expectorating the pneumonic products. When, despite this, edema of the lungs supervenes, as shown by the loud mucous bubbling rales over the pneumonic and other areas of the chest, the chances for success become less, but even then I have the attendant regularly rouse the patient to coughing efforts as long as consciousnes is retained. As far as one may judge, stimulation in acute pulmonary edema occasionally keeps the patient alive for some hours or even a day, and may exceedingly rarely seem to pull a patient from death.

With lytic or gradual resolution, even with very sharp critical drops of temperature, the battle is usually won, for edema of the lungs rarely occurs, and stimulation may be efficacious. However, even in cases of non-massive pneumonic involvement in young, vigorous individuals, with no evidence of toxicity during the attack, sudden, almost tragic intrusion of critical resolution, edema of the lungs, and death—all within a few hours—may supervene. One instance especially impressed me—that of a young, healthy individual to whom the pneumonia was literally a laughing matter. From the presence of sudden perspiration and from the physical signs in the lungs, I was able to foretell the onset of critical resolution some hours before the typical drop in

temperature. Heart and pulse were then perfectly satisfactory. Herculean efforts were made by stimulation and otherwise to ward off the dangers of edema of the lungs. Within six hours, however, the patient died from pulmonary edema; while in an adjoining ward, an old woman, severely poisoned by her pneumonia and very sick for over one week, had reached the crisis with slow, gradual resolution of the pneumonia, and slowly but surely convalesced.

Where digitalis has been given, its effect in causing arrhythmia must naturally be considered. Aside from this, a study of cardiac irregularities occurring during the course and at the crisis of pneumonia offers interesting and important therapeutic problems. The usual types are moderate bradycardia, sinus arrhythmia, extrasystoles, auricular fibrillation and heart block. It is sometimes assumed that the presence of such arrhythmias in pneumonia is indicative of some organic affection of the cardiac valves or musculature. But, as in the non-pneumonic individual, irregular heart action does not in itself necessarily mean heart disease. Indeed, some of the arrhythmias—especially sinus arrhythmia, moderate bradycardia and extrasystoles—when found with an unembarrassed circulation at or immediately after the crisis usually offer good clinical evidence that the disease has definitely run its course. The irregular cardiac action is apparently due to a neurotropic effect of pneumonic toxins. Whether the latter act upon the medullary center or upon the heart itself it is impossible to state. Because of the occurrence of these cardiac irregularities at the time of crisis, and because some drugs (for example, morphine) produce arrhythmias by affecting the cardio-inhibitory center, I am inclined to the view that these arrhythmias are due to an effect on the cardio-inhibitory center rather than on the heart itself. These arrhythmias usually last a few hours or days. I have followed some of these cases for years and have never observed any correlation between the arrhythmias and the possible later development of organic cardio-vascular disease.

I have observed auricular fibrillation—complete irregularity of the pulse—several times at the crisis. In three such patients, each of whom had several crises from successive involvement and resolution of various pulmonary areas, there were attacks of fibrillation lasting several hours with each crisis. Two of the patients were elderly; the third, a vigorous adult. None showed circulatory embarrassment or heart failure during the course of the fibrillation; on the contrary, there was the usual picture seen in favorable crises with normal rhythm. On the other hand, I have observed other cases of auricular fibrillation occurring during the course of toxic pneumonia, all of whom died. In these it was impossible to estimate how much the arrhythmia itself contributed to the dangers of the pneumonia.

Complete heart block with slow ventricular rhythm is rarely found in pneumonia. That it may be functional in origin and probably due to abnormal action upon the cardio-inhibitory center seems indicated by two of

my cases, one of which came to necropsy. The patient had been ill for some time, there was no history of digitalis medication. Microscopically and grossly, the cardiac musculature and the bundle of His were found normal.

The therapeusis of arrhythmias developing in pneumonia is similar to that in non-pneumonic individuals. If accompanied by heart failure, the arrhythmias are of serious, possibly ominous, import. Except in heart block with slow ventricular activity, when atropine should be tried, the treatment of these arrhythmias is no different from that of the other phases of pneumonia already discussed. If arrhythmias are unaccompanied by failing circulation, though they perhaps should not be entirely disregarded, their import is slight and they rarely require separate medication.

It may perhaps emphasize my standpoint on cardiac stimulation in pneumonia to summarize my procedure in the circulatory treatment of pneumonia in the influenza pandemic of 1918–19, when pneumonias were seen early and the therapeutic course could be followed from the very inception of the disease. As stated, the important therapeutic effort lies in cardiac stimulation. I shall merely allude to the fact that numerous plans were then in vogue, regarding the dosage, time of administration and kind of stimulation to be employed. My practice has been in this and other epidemics to begin stimulation early in severe cases, or in those likely to become severe. I digitalize early because the fight is usually a short one—the patient recovers or dies within a few days. I have never found any untoward effects upon the heart, kidneys, pulse rapidity or blood pressure from this plan, nor have I found that the use of any special digitalis preparation had produced any demonstrable difference. When available, and the patient is able to swallow, I prefer the standardized digitan tablets. When not available, I have used the tincture of digitalis. When I wished a quick effect, I administered the latter in 60-minim doses every few hours; when not so urgently indicated, I gave it in 15-minim doses every four hours. I believe that the criticism of the variable strength of the tincture is valid; but when the drug is employed in large doses and frequently, I feel that, gauged by the usual amounts required to digitalize in cardiac disease—(from one to one and one half ounces) the pneumonic patient soon receives enough to properly digitalize him. In exceptionally severe cases I added a hypodermic of strophanthin in the ampule of $\frac{1}{1000}$ strength, even after the patient had already received much digitalis. I have seen neither bad nor good results from this procedure, although I believe the procedure hazardous in ordinary cardiac disease. I used caffein benzoate of soda liberally, usually in hypodermic form. The average dose was 5 grains of the double salt in solution, given every two to four hours, depending on the severity of the case. I observed the use of camphorated oil in cases in which I was consulted. I do not believe this drug possesses any chemotherapeutic or stimulatory power, despite its immense popularity in previous years. Phlebotomy I also employed in a few cases. I believe its chief value lies in getting rid of toxic material. I saw no beneficial results follow its use in the epidemic.

I employed adrenalin often, but with no effect. In edema of the lungs, I used atropin and dry cupping of the chest, and crowded stimulation. These procedures had no demonstrable effect in preventing or even retarding a fatal termination. Where indicated I used the usual expectorants—ammonium chloride and carbonate, the iodides—without demonstrable effect.

I have sketched my "plan" of therapy and stimulation in some detail because I wished to demonstrate that I have followed the usual well-known and well-worn methods. I confess, however, that I could observe no beneficial result from the use of stimulation in the pneumonia pandemic. I may also add that for many years it has been my contention that the almost innumerable and varied therapeutic "plans," many of which have already been discarded, have not met with any demonstrable success in my hands, for I was never able to correlate the therapy with any effect upon the circulation. The milder cases require no stimulation. Some of the severe lobar pneumonia cases did get well, it is true, when stimulation of various kinds was used, but I am not convinced that it was the stimulation, and not the specific types of virulence present, that saved these cases.

In personal conversations with many practitioners and consultants, the great majority acknowledged that they were therapeutically helpless in the pandemic. And, may I not add, has stimulation helped the circulation in other types of pneumonia? May we not be compelled to radically revise our views on what stimulation can actually accomplish?

Bearing in mind the clinical, experimental and pathological data, may we not find therein sufficient cause for heart failure and death, and the comparative futility of cardiac stimulation in pneumonia? Can cardiac stimulation of any kind, no matter when begun or how continued, counteract pneumonic chemical poisons which are being continually elaborated throughout the disease, and which at certain times—the crises—are elaborated rapidly and in great amounts? This appears to me the fundamental problem of cardiac stimulation in pneumonia. I believe we must possess, in addition to stimulation, serological or chemotherapeutic methods for counteracting or nullifying the pneumonic toxins, or some method of changing a rapid into a slow resolution. Perhaps even a mechanical method for literally aspirating pneumonic products, by suction or otherwise, is not impossible in these days of advance in tracheal insufflation. The heart can apparently withstand a certain amount of toxins, moderate in quantity, which are *slowly*, not suddenly, thrown into the circulation. Elimination and antibodies can then perhaps keep pace with production of toxin. Otherwise cardiac power is slowly or quickly weakened, and cardiac stimulation is unfortunately usually futile.

Having described in detail circulatory disturbances and the circulatory treatment of pneumonia, it remains to emphasize that the functional power of the heart is not only seriously interfered with during pneumonia, but that actual myocardial degeneration, more serious than the common cloudy

swelling (Chapter V) can occur. Finally I wish to give a few illustrative cases, demonstrating severe cardio-sclerosis as a *late* sequel of pneumonia. The manner in which the myocardium is damaged in pneumonia is still in dispute. I am of the opinion that the myocardial damage is usually the result of the toxemia. This is to some extent based upon the observation of two cases of heart block that occurred during pneumonia. From the first, a man of 70, I obtained typical polygraphic tracings of heart block. The arrhythmia was not accompanied by special signs of circulatory failure. An autopsy was obtained. Macroscopic examination of the brain and spinal cord, and microscopic examination of the heart, including the junctional tissues, showed that all these structures were normal. The second case, a man of 50, entered the hospital in collapse and semi-stupor. The temperature was 104°. There was pneumonic consolidation of the left upper lobe. The temperature ranged between 100° and 104° for one week. The heart sounds were scarcely audible, the radial pulse was regular, its rate between 100 and 120 per minute. At the end of one week there was critical defervescence, the temperature fell to 97.4° per rectum, there was extreme collapse. The ventricular rate suddenly dropped to 44, and remained between 30 and 44 for four days. Although satisfactory polygraphic records could not be obtained, heart block was diagnosed by the clinical phenomena and pulse rate. The pneumonic area underwent gradual resolution. There were no general convulsions, but frequent convulsive tremors of the musculature of the upper and lower extremities. The stupor slowly deepened into coma. Suddenly, one week after the inception of the slow rhythm, the patient sat up in bed fully oriented, with good color, warm extremities, and a pulse and ventricular rate of 76 per minute. The heart sounds, though still faint, were readily heard. Except for two occasions a few days later, the pulse continued regular. The patient returned some weeks later with edema of the lower extremities, apparently due to myocardial changes.

The heart block in the first case may have been due to an effect of the pneumonic toxins upon the cardio-inhibitory center. At any rate, since the autopsy revealed a perfectly normal heart, it demonstrated that toxins may have only a temporary effect upon the cardiac musculature, and that the latter may then again return to normal. In the second case, the inception of the heart block was accompanied by circulatory collapse; the latter disappeared with return of normal rhythm. Several weeks later there was again mild heart failure. The pathological sequence would seem to have been heart block and collapse at the crisis, partly from a toxic inhibitory effect and partly from severe acute poisoning of the myocardium. From this attack the patient recovered; this was followed later by sufficient myocarditis to cause mild decompensation.

**Cardio-sclerosis as a Late Sequel of Pneumonia.**—Damage to the cardio-vascular apparatus as a *late* sequel of pneumonic infections has been insufficiently emphasized. Such sequelæ produce symptoms only some

months or years after the pneumonia has run its course, so that the connection between the two diseases is usually entirely overlooked. In some instances the pneumonic poison seems to light up a dormant cardio-vascular process; in others, it primarily produces this condition. Brief case reports of both types follow:

Male, age 50, entered the hospital with a mild pneumonia. He recovered within one week. The only point of interest in a careful examination of the cardio-vascular system was a slightly accentuated second sound over the right base suggestive of aortitis. The Wassermann blood reaction was negative. The urine and blood pressure were normal. Two months later the patient re-entered the hospital with the history of a pneumonic attack three weeks previously. He presented all the typical evidence of cardio-sclerosis with decompensation—anasarca, urine containing albumen and casts, high blood pressure, markedly accentuated second aortic sound. Evidences of the recent pneumonia were still present.

A physician, age 50, had for several years remarked occasional dyspnœa upon walking but never considered himself ill. Two years before coming under observation, he developed a severe pneumonia which ran a toxic course and lasted several weeks. A few months thereafter, cardiac symptoms began. He became dyspnoic and edematous. Hypertension developed. The urine contained albumen and casts. Orthodiascopic examination one year later showed marked enlargement of the aortic arch and of the left ventricle. With some remissions, the cardiac symptoms lasted until the time of his death, one year and a half later.

Both these cases illustrate the effect of pneumonia upon what seemed otherwise quiescent cardio-vascular disease.

Male, aged 32, never drank or smoked. He had a grippe infection with fever six years previously; this lasted several months; pulmonary tuberculosis was suspected but bacilli were never found. Cardio-nephritic symptoms began three years later. They were at first mild and consisted of occasional dyspnœa when climbing stairs. The signs of cardio-sclerosis gradually progressed so that at the time of examination, although the patient complained of but slight dyspnœa and precordial pain, the left half of the chest was practically filled by a hugely hypertrophied heart. The systolic blood pressure ranged between 200 and 300 m.m. of mercury, the first sounds at the right base and at the apex were exceedingly harsh, rough and vibrant, indicative of probable calcification of the mitral valves and aorta. The second sound at the right base was markedly accentuated; the urine contained albumen and casts. The Wassermann blood reaction was negative. The clinical picture was that of extremely advanced cardio-sclerosis and nephritis.

From the history and from the absence of other etiological factors, it is fair to conclude that the prior pulmonary infection six years ago was the direct and only cause of the extreme cardio-vascular disease.

## CHAPTER XXVIII

## CARDIAC DISEASE IN MARRIAGE AND PREGNANCY

The question of marriage in women with cardiac disease is one which evidently requires careful consideration. The chief dangers arising from marriage in such cases are twofold; The strain and excitement incidental to coitus, and the dangers arising from pregnancy. Regarding the former, I know of several instances of hemoptyses immediately following coitus in patients with mitral stenosis and auricular fibrillation (complete irregularity of the pulse), and in one with cardio-sclerosis and hypertension. I also know of one case of death from cerebral apoplexy following intercourse; the patient was a man with cardio-sclerosis and hypertension. I have observed cases of violent tachycardia caused by intercourse. With two exceptions, the cardiac condition of all of these patients would have been considered fairly satisfactory under ordinary conditions.

Views regarding the advisability of permitting marriage in women with cardiac disease are quite at variance. For example, some maintain that patients with heart disease should never marry, no matter what type of lesion be present. I believe this view to be extreme. One frequently encounters mothers of large families who doubtless have had valvular disease for many years and who have gone safely and normally through pregnancy and parturition. Many of these patients were never aware of their disease; in some the lesion was discovered only in the course of a routine examination.

The statistical tables of the lying-in institutions that I have studied have, in my opinion, very little value in clarifying the problem. The statistics are based chiefly upon patients that entered the hospitals severely decompensated; a cardiac history prior to pregnancy or to decompensation is entirely disregarded or only cursorily mentioned. To be of value, cases must be carefully studied from the cardiac as well as from the obstetrical standpoint; this requires the cooperation of internist and obstetrician, or the skilled observation of that rare combination, a clinician who has had a large, active obstetrical experience.

Marriage naturally should not be advised, if at the time the patient shows the slightest degree of decompensation. Briefly, decompensation is evidenced by the presence of dyspnœa, especially on exertion; by edema of the extremities; an enlarged liver; bronchitis; cyanosis, and by arrhythmias. The latter are chiefly extrasystoles (pulsus bigeminus), auricular fibrillation (complete irregularity of the pulse) and tachycardia. I wish to emphasize, however, that arrhythmias in themselves, except possibly auricular fibrillation, do not

necessarily indicate either heart disease or decompensation (Chapter XI). In those who have only recently recovered from heart failure, it is a safe rule to interdict marriage until at least two years have passed without subsequent break in compensation. An exception is noted later with reference to aortic lesions and extreme cardiac hypertrophy. The interval mentioned—two years—is, of course, purely arbitrary, but seems to agree best with clinical experience. In valvular lesions the same time should be set as a safe interval in which no inflammatory symptoms have occurred. If, for example, there have been rheumatic endocarditic recrudescences and recurrences, as evidenced by louder murmurs, slight febrile attacks, pericarditis, tachycardia, arrhythmias, or other clinical manifestations, marriage should not be advised, for the chances are in favor of another attack within one year. In short, marriage (and pregnancy) may be considered safe if excellent compensation and freedom from endocardial exacerbations persist during the two-year period. The physician who has not had the opportunity to observe the patient within the prescribed antenuptial period must necessarily be guided by the history and physical signs.

Of the two factors, decompensation and quiescence of the lesion, I believe that the latter is the more important. In my experience, more danger and more fatalities have resulted in pregnancies from marriage occurring when lesions were active than from mild decompensation. Gestation seems to light up dormant or only partially active cardiac processes.

Cardiac symptoms frequently begin early in pregnancy, sometimes even in the third month. At the beginning, simple pulse acceleration occurring in attacks (paroxysmal tachycardia) may be present. Slight fever may appear; when due to endocardial exacerbations, it is of serious import. The occurrence of new or louder valvular murmurs, or of fresh pericarditis, may furnish clues of such recrudescences. Hemoptysis is not infrequent. As pregnancy advances, unless the above or similar manifestations recede or are checked, dyspnœa and cyanosis gradually supervene, and with them the usual symptomatology of frank cardiac decompensation; edema, orthopnœa, enlarged liver, pulmonary congestion, arrhythmias (especially extrasystoles), etc. The greater circulatory demands made by the growing placental and fetal circulation very probably also play a role in causing this heart failure. Labor, induced or spontaneous, does not always terminate the circulatory embarrassment, for insidious endocarditis may continue and death result from some complication or from circulatory failure.

The above considerations refer to marriage in women with all types of valvular lesions. It remains to differentiate between these on the basis of clinical experience. I have found that rheumatic mitral stenotic lesions are the most dangerous during pregnancy. The patients readily develop paroxysmal or, more often, simple tachycardia, which may last during the greater part of pregnancy. The rapid heart action itself may produce such dyspnœa or discomfort that the induction of premature labor is indicated.

Hemoptyses are common. Bronchitis with sibilant breathing and mucous rales over the entire chest is not unusual. As occasional complications during the puerperium, one may mention embolic infarcts in the lower extremities or in the lungs.

The history of pregnant women with mitral stenosis and auricular fibrillation presents a varied clinical picture. Some of these patients date their first break in compensation at a first or second pregnancy which had been carried to full term. On the other hand, I have observed patients who went through successive pregnancies with mitral stenosis and auricular fibrillation, with no cardiac complications or symptoms. One of these deserves brief mention; when first examined, she was fifty years old, with general anasarca, orthopnea, auricular fibrillation, a double mitral lesion and an old rheumatic history. She had had eighteen children without cardiac symptoms. The latter began only three years after her menopause, which commenced when the patient was forty-five years old. From her history, it seemed probable that fibrillation had been present at least during her later pregnancies. Another patient, 53 years of age, was the mother of five children. She had a rheumatic history and double mitral lesion with auricular fibrillation for many years. During her pregnancies, the cardiac symptoms were very slight and of the same nature as those occurring when she was not pregnant; these consisted in occasional dyspnœa and tachycardia.

Except by means of the general considerations above outlined, there seems no way of determining in advance the favorable or unfavorable subjects for pregnancy, in those with auricular fibrillation. Extreme caution in advising marriage or pregnancy is, of course, necessary because of the known tendency of patients with this arrhythmia to decompensate.

Patients with simple mitral regurgitant lesions are the most favorable subjects for pregnancy and the most apt to go through gestation without untoward cardiac complications. When the latter do occur, they are more often of the mild decompensatory type from recurrence of the endocarditis.

Pregnant women with aortic lesions suffer chiefly from tachycardia. This is true of those with, as well as those without, marked ventricular hypertrophy. In the former, however, tachycardial attacks occur more frequently, are more readily evoked and are of longer duration. Decompensation is comparatively rare in those with but moderate or slight hypertrophy. When extreme ventricular hypertrophy exists, cardiac failure is apt to occur early in pregnancy, a tendency increased by the rapid heart action. Such patients should be advised against marriage or, if already married, should not be permitted to become pregnant even if the lesion is quiescent, and compensation in the nonpregnant state is good.

In all types of decompensated endocardial lesions occurring during pregnancy, the question of the induction of abortion or of premature labor arises. Severe cardiac failure in early pregnancy (before the fourth month) or slight decompensation which does not yield to treatment is, I believe, an indication

for immediate emptying of the uterus. This indication is not vitiated by the fact that some of these mothers may, by protracted rest and medication, carry the child to viability or even to full term without further complications. The life of the mother is the prime consideration and should not be jeopardized, as it would be, in an attempt to continue the pregnancy when cardiac decompensation is present in early gestation. My observation has been that an abortion, surgically clean and skillfully performed, is only slightly, if at all, more dangerous in cardiac patients than if performed for other reasons on those with normal hearts. I am also strongly in favor of terminating an early pregnancy if it is evident that there exists recurring endocarditis. As already stated, this may consist in the presence of fever, in the onset of paroxysmal or constant tachycardia, of extrasystoles or other arrhythmias, of frequent hemoptyses, and of changes in the physical signs.

If the signs of decompensation or of fresh endocarditis appear between the end of the fourth month and the time of viability, in view of the somewhat more serious operative procedure required to induce miscarriage, the decision regarding interruption of gestation hinges chiefly upon the severity of the cardiac complications. If decompensation is mild, or if the evidence of fresh endocarditis is not severe, appropriate therapy should at first be attempted for about a week or ten days. Should the symptoms then disappear and the patient improve, pregnancy may be allowed to proceed until the period of viability, possibly until normal labor. If decompensation or endocarditis does not react well to therapy or becomes suddenly severe or threatening, it is much safer to induce miscarriage.

In the interim between the seventh and one half and the ninth months of gestation—the period of viability—the decision regarding the interruption of pregnancy in decompensated cases or in those with recrudescent endocardial lesions is of less vital importance because the premature induction of labor in proper hands adds scarcely any risks. The question of waiting a month or more until the more natural process of normal labor occurs must depend upon the cardiac condition; that is, if there is any reason to fear the slightest increase of cardiac complications, it is both wiser and safer to have pregnancy terminated soon than to wait until full term.

There are two gynecological methods employed to end labor when the child is viable: One, the introduction of bags into the uterus; two, Cæserian section. The first method usually produces a tedious and often an abnormal labor, so that the patient's already diminished cardiac reserve may become considerably decreased by long continued pains and the length of time required for the process of parturition.

Regarding the general question of cardiac strain incident to pregnancy and labor, much has been written, but on the whole we possess no accurate information or knowledge of many factors here involved. Undoubtedly the demands made by the growing fetal circulation; interference with venous return in the splanchnic area by the growing uterus; gradual invasion of free

diaphragmatic mobility by the uterus and by displaced abdominal contents are physical factors of importance during pregnancy in those with diseased hearts. Added to these is the cardiac strain incidental to labor itself. Bearing down expulsion pains unquestionably call for more cardiac energy and hence cause cardiac strain both by producing venous congestion and by holding the breath during the labor pains. Such efforts may finally result in more or less continuous dyspnœa. Prolonged labor—dry labor, for example—by interfering with sleep and by keeping the patient's nervous condition keyed up and on edge, may gradually interfere with and decrease an already diminished cardiac reserve, so that more or less obvious signs of mild heart failure, especially dyspnœa and tachycardia may finally eventuate. In addition, the obstetrician can rarely give definite information, especially in primiparæ, as to whether the labor is going to be difficult or easy, quick or slow. All these questions, some answerable, others not—are intimately bound up with the method of procedure to be followed during labor, and more particularly, at the stage of viability. At full term, labor should naturally be shortened and made as easy as possible for the patient. Pituitrin, when indicated in those with normal hearts is also indicated in those with heart lesions. Forceps should be applied as soon as the procedure is considered safe.

With reference to induction of labor at the stage of viability, the choice between divulsion with bags and Cæserian section arises. The former is very apt to give rise to a tedious labor with all the disadvantages from the cardiac standpoint just alluded to. Regarding Cæserian section it is perhaps surprising to observe how well women with heart disease withstand the operation. My personal experience has been limited to two cases, both with mitral regurgitant lesions. One had moderate dyspnœa during the last two months; the other had dyspnœa during the last month of pregnancy. In the latter the dyspnœa responded quickly to rapid digitalization, so that at the time of operation, compensation was fully restored. After Cæserian section in the two cases, the cardiac condition was satisfactory during and immediately after the operation. There was no untoward rise of the pulse rate. Dyspnœa, the main symptom, disappeared within 24 hours. The succeeding days showed a steady improvement; indeed, the heart reacted no differently than after any skillfully performed Cæserian section in those with normal hearts. Of interest is the previous cardiac history of one of these cases. This Cæserian section was her third pregnancy. Her second labor lasted one week. She stated that it took months for her heart to finally recuperate so that she could walk comfortably and go about her household duties. The last time I saw the patient was two weeks after the Cæserian section. The cardiac rate was normal. She felt perfectly well with the single exception that she could not yet lie comfortably on her left side. From all indications she will very probably recuperate perfectly from the cardiac standpoint by the time she is allowed out of bed, for it is the laparotomy, not the heart,

which keeps her in bed at present. This is an instance where the operation not only saved her the cardiac strain of labor but also its annoying, long continued, after effects.

One word of caution is necessary regarding Cæserian section and that is that compensation should be restored as much and as quickly as possible by thorough digitalization before operation.

An addition to the causes already given which may interfere with and complicate pregnancy in women with cardiac disease is superadded cardiac neurosis.

Patients with various type of perfectly compensated valvular diseases and with no new outbreaks of endocarditis for years, may, because of their knowledge of the cardiac disease and because of their dread of childbirth, present cardiac symptoms of a neurotic type which in themselves may call for interruption of pregnancy. Two such cases are epitomized:

Mrs. K., aged 21, married one year and now pregnant three months, has had a rheumatic double aortic lesion for years. In the past ten years her only complaint has been occasional palpitation due to moderate tachycardia. Since her pregnancy, she has had frequent crying spells because obsessed with the idea that she would die during childbirth. She complained almost continuously of palpitation. Examination revealed, besides the valvular lesion, moderate left ventricular hypertrophy, and a cardiac rate of over 100 per minute. Compensation was perfect. Because of her nervous condition, the induction of miscarriage was advised. She was told, however, that if, at a subsequent pregnancy, she were less nervous, she might carry the child to full term with no danger to her heart. Curettage was performed under gas and ether without untoward symptoms. Within one year of the operation she again became pregnant. There were no neurotic or cardiac symptoms, no tachycardia or palpitation. She carried the pregnancy to full term, the delivery and puerperium were normal.

Mrs. L., married two years, is now pregnant six weeks. She has a typical double mitral lesion. Despite the valvular disease, she was always active as a child and has never suffered from decompensation. She is of a very neurotic temperament and is easily frightened. Under such circumstances she suffers from palpitation which, on examination, is found to be due to rapid heart action, usually at the rate of 110 per minute. When first examined, I advised her that the continuation of the pregnancy would depend more upon her general nervous condition than upon her heart, that the former affected the latter, and that if she were not nervous and frightened, there would be no reason to worry about the condition of her heart; her heart was in as good condition as it was ever likely to be. Mixed bromides were given. Ten days later she again consulted me. She feared she would lose her life if pregnancy continued. She had been having sleepless nights and crying spells. Palpitation had been frequent, almost constant. I therefore advised immediate termination of pregnancy, though compensation was

perfect. In this case I also felt that a future pregnancy might be carried to full term without mishap if the patient did not become frightened and nervous. Two years later she again became pregnant. She was delivered of a healthy child by Cæserian section. Convalescence was normal. I again saw her one year after delivery. Her cardiac condition was satisfactory, compensation good.

The medical treatment of the various cardiac symptoms occurring during pregnancy may now be briefly sketched. Decompensation, no matter whether evidenced by circulatory congestion (enlarged liver, edema, bronchitis, dyspnœa, cyanosis) or by arrhythmia calls chiefly for digitalis and rest. I believe that patients should be brought under the influence of the drug as quickly as possible. Where cardiac failure is extreme, the case urgent and digitalis has not previously been given, it is best to begin with the intravenous or intramuscular injection of strophanthin (dispensed in ampules in a $\frac{1}{1000}$ solution); the dose is 10–15 minims. When given intravenously, it should be injected slowly. After twelve to twenty-four hours, digitalis in the usual doses can be given. I am in the habit of prescribing the tincture in 15-minim doses three or four times daily, given undiluted. The average amount for a full effect is one ounce, distributed over a period of about one week. When dyspnœa or restlessness is marked or pulmonary edema is present, luminal tablets, one and one-half grains each once or twice daily, or occasional doses of morphine are indicated. Special diet is sometimes of value in getting rid of edema. Where the urine output is normal in amount, I have found restriction of fluid intake in twenty-four hours to 1000 c.m. of milk (Karrell diet), or of that amount or even considerably less of other fluids (*e.g.*, water, lemonade, weak tea) for two or three days in succession, combined with the administration of theobromine-sodium salicylate in $7\frac{1}{2}$-grain doses three or four times daily of decided advantage. To appease the hunger pangs, when present, dry toast or baked apples may be added.

For the treatment of endocarditic recrudescences the salicylates are required; they should be administered up to their physiological limit. I usually give the salicylate of soda in one gram doses until eight doses have been given or until tinnitus occurs. The dose is then decreased or medication temporarily discontinued. I have found no beneficial results from serums, vaccines or from preparations of silver salts. Unfortunately, salicylates are sometimes of little or no value. Ice bags occasionally control rapid heart action. They are to be discontinued if no effect follows their use or if they make the patient uncomfortable.

The chief types of irregularities have been mentioned. If arrhythmia is the result of decompensation, digitalis should be given in the usual manner. If due to fresh endocarditis, besides the salicylates, bromides in large doses or luminal may be of value, especially in the presence of tachycardia. Arrhythmias (usually extrasystoles), which are occasionally caused by digitalis soon after its administration has been begun, do not contraindicate its further

use if the object of the medication—the restoration of compensation—has not been accomplished.

**Summary.**—We possess at present no instrumental guide or tolerance test which can adequately measure the cardiac reserve of the diseased heart of the pregnant woman. Each case must be individualized and the presence or absence of heart failure noted. The best clinical guide for heart failure is a detailed study of the patient's symptoms together with a detailed study of the physical signs. Cardiac neurosis, decompensation and endocarditic recurrences seem the best inclusive groupings of all types of cardiac symptoms that occur in pregnancy with heart disease. The degree of decompensation and the month of pregnancy together determine the type of procedure. Decompensation that does not react to medication quickly or permanently, requires interruption of pregnancy at any stage. When viability has been reached and decompensation is moderate or absent, Cæserian section, skillfully and rapidly performed, seems to be well withstood by the diseased hearts of pregnant women. It prevents the cardiac strain incidental to labor; it may likewise prevent a lingering, cardiac recuperation following labor. Should the woman come to term the labor should be shortened as much as possible. Ether is the anesthetic of choice.

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## CHAPTER XXIX

## CARDIO-VASCULAR EXAMINATIONS OF WAR RECRUITS

This subject is of importance because it indicates the types of young men seen in routine clinical work.

Men of the draft age, that is those between twenty-one and thirty years of age, who present themselves for examination before the local draft boards may be divided into two groups, in regard to cardio-vascular conditions. (1) Those with frank, unmistakable cardio-vascular disease; (2) those with cardiac abnormalities, that is, with physical or clinical signs which resemble and may be mistaken for real cardio-vascular disease.

1. **Evident Cardio-vascular Disease.**—By far the larger percentage of candidates in this category have valvular lesions; a much smaller group comprises those with hypertensive cardio-vascular or cardio-renal disease. Myocarditis as an entity in the adult of the draft age need scarcely be considered because its incidence is negligible, and its discovery and diagnosis as a single pure lesion are surrounded by pitfalls and mistakes (Chapter VII). It is self-evident that men having valvular disease with signs of advanced or even beginning decompensation are not fit to become soldiers and hence should be rejected. It may become a part of the medicomilitary problem properly to sort the valvular cases which are perfectly compensated and present no symptoms and who even if non-combatants can be of some military service. I have met men, for example, who knew of their lesions and yet were sufficiently patriotic to wish to enter the army in any capacity. One such case was that of a young lawyer, twenty-three years of age, who had mitral regurgitation for ten years and who in order to prove to himself his capacity for marching and for exertion, had taken a fifteen mile hike. Examination by me the next day revealed no ill effects from the walk. I also recently had the opportunity to examine two infantry recruits in one of the cantonments, both of whom had been in camp several weeks, and whose lesions were discovered only while making a routine chest examination. It then appeared that both of the men had known of their valvular disease; both had succeeded in convincing the examining physicians of the local draft boards that they had only heart murmurs and not heart disease; both stood the preliminary training without mishap or symptoms; both were exceedingly anxious to stay in the service. While no special provision had been made for perfectly compensated cases of this type, it would seem unwise to put them, even if willing, in such laborious service as is entailed in the infantry or engineering. The probabilities seem more in favor in their decompensating or of their developing the irritable soldier's heart (Chapter XVIII), with its tachycardia and other

symptoms than will those with perfectly normal hearts. Of perhaps equal importance is the possibility that trench and other disesaes—trench fever, trench nephritis, catarrhal fevers, pyrexias of unknown origin, or pneumonias —may light up dormant and quiescent endocarditic lesions with resultant syndromes similar to, or as serious as, the usual recrudescences of valvular disease. With cooperation between the medical and the military branches, there appears no harm in admitting these perfectly compensated and quiescent valvular cases into the less laborious work of the quartermaster's or similar departments.

Hypertension for the purposes of draft examination I would define roughly as a systolic blood pressure of 150 m.m. or over. Blood pressure estimations have thus far not been done as routine procedures in the local boards. Associated with the presence of albumin and casts in the urine, men with hypertension, even if moderate, should be regarded as unfit for combatant military service. Another group with hypertension is that found in recruits with tachycardia. The desirability of accepting these recruits depends not upon the hypertension, even if considerable, but upon the tachycardia, a subject which will be discussed later. Still another group, though small, consists of those with moderate hypertension but with no evidence of renal or cardiac disease, cases of functional hypertension (Chapter XXV). These comprise the cases which in civil practice are usually rejected for life insurance. Repeated examination of the urine and of the heart shows no renal or cardiac disease. I feel that these individuals represent hypertension, temporary or permanent, due to emotional excitement incidental to the examination, or to heightened vasomotor tone from obscure though unimportant reflex causes. I believe that when careful and repeated examination of the urine and heart shows no abnormality, and the usual symptoms accompanying hypertensive cardiac disease are absent, the applicants should be accepted in the ordinary routine fashion. In civil practice, where I have been able to follow similar cases for some time, the young men have been athletic and have followed their vocations without hindrance or untoward symptoms.

Another temporary cause of hypertension which is usually overlooked is that due to abnormal resistance of the arterial wall, known as arterial tonus (Chapter XXV). This cause can be readily determined by keeping up the cuff pressure on the brachial for a few minutes before a second systolic reading is taken. When the hypertension is due to hypertonus, the second reading will be within normal limits and will represent the actual blood pressure of the individual. The second systolic reading is sometimes 15 to 30 m.m. lower than the first.

**2. Signs Simulating Cardio-vascular Disease.**—We now come to a larger and more important group of applicants, *viz.*, those with physical or clinical signs simulating actual cardio-vascular disease. All these may be regarded as non-organic (functional) in nature; hence, with the limitations to be mentioned, such recruits may be drafted in the service. For purposes of classifi-

cation, this subject may be studied from the standpoints of: (1), size of the heart; (2), abnormal cardiac rhythm; (3), abnormal cardiac sounds, and, (4), dyspnœa.

1. **Size of Heart.**—In another chapter (Chapter XII), I have indicated the common sources of error inherent in percussion methods which attempt to delimit the cardiac area with any exactness. Frank and gross enlargements can be readily diagnosed, but their importance lies in the category of those with definite lesions. I consider it of minor importance in those with equivocal physical signs of cardiac disease to be able to map out the exact size of the heart, for there are many factors which produce considerable variations even within normal limits. The broad and deep chested are apt to have larger hearts than the small and narrow chested (Chapter XII). The tall and gaunt have as a rule, narrow, graceful and pendulous hearts. The fat and stocky are apt to have hearts which tend to occupy horizontal positions in the chest. There are, however, numerous exceptions. For example I have often been surprised to find upon fluoroscopic examination that thin and narrow chested individuals possessed large, though normal, hearts. Fluoroscopy will also confirm the fact that the actual apex is often several centimeters below the point of the maximal apical impulse. To those variations as found in quiet, normally beating hearts, I may further state that hearts beating vigorously and rapidly usually give the impression of enlargement, although X-ray examination often reveals them to be of normal contour and size. These brief considerations show the difficulty of standardizing the size of the heart and of mapping out its size and contour by percussion. However, while for other purposes such exact knowledge may be of interest and importance, it possesses but slight and subsidiary value in the examination of recruits with hearts simulating actual disease.

2. **Abnormal Cardiac Rhythm.**—The great majority of cases in this group are those with simple pulse acceleration, usually due to the incidental, emotional excitement of the examination. The usual rates are in the neighborhood of 100 a minute. Sinus arrhythmia, *i.e.*, an alternate slowing and acceleration of the cardiac and pulse rate in consonance with inspiration and expiration, respectively, is less frequent than simple tachycardia. A combination of slow and then rather rapid heart action, with sudden transition from one to the other, each phase lasting approximately four to eight beats, is not unusual. Occasionally premature contractions (extrasystoles) are encountered. Tachycardias of paroxysmal nature, that is, sudden attacks of extremely rapid cardiac acceleration with ventricular rates between 150 and 200, lasting several seconds or minutes, are occasionally found in the draft candidates. All of these cardiac irregularities are readily diagnosable by ordinary clinical methods. Of even more importance than their diagnosis is the fact that none of these irregularities are in themselves indicative of, nor do they form any basis for, the diagnosis of heart disease (Chapter XI). They are usually of purely functional origin. Other concomitant signs and

symptoms in addition to cardiac irregularity are necessary before the candidate can be classified as having cardiac disease. A persistent tachycardia, however, may produce sufficient subjective annoyance and objective dyspnea upon exertion to unfit the candidate for the duties of the soldier. Such men, to be finally rejected, should present this symptom while under observation for at least one-half hour, the rates should be 120 or over, the tachycardia should be accompanied by unmistakable evidences of dyspnea, and the same pulse acceleration should again be present at some future examination.

3. **Abnormal Cardiac Sounds.**—I refer here to those murmurs, reduplicated (so-called gallop and double rhythms) and other adventitious sounds heard over the normal heart. All these may be conveniently grouped as occurring over the right base, over the left base, or over the apical region. Over the right base are heard soft, whiff-like systolic murmurs usually accompanying, not replacing the normal first sound. They commonly occupy the whole systolic period. They are rarely transmitted to the carotids, although undue pressure with the stethoscope upon these vessels may give that mistaken impression. Unless the definite signs of an aortic stenotic lesion are present, the murmur just described is not significant of actual disease.

Over the left base are found chiefly the so-called cardio-respiratory murmurs. These have a superficial character, may be quite loud, and are usually systolic, though very rarely both systolic and diastolic in time. The murmur is sometimes transmitted to the midprecordium or even lower. Its intensity usually varies with the position of the patient and with the phase of respiration. It is ordinarily least intense and indeed may be found to disappear entirely if auscultation is practiced at the end of deep inspiration. Even if loud, these left basal murmurs are not indicative of heart disease. They are to be differentiated particularly from the friction sounds of localized pleuropericarditis in this area, as well as from patent ductus arteriosus and pulmonary stenosis. In the latter cardiac lesions, there are definite physical signs and symptoms (Chapter XIX).

Different from murmurs over the left base, are the reduplicated sounds, usually called gallop or double rhythm (Chapter XIII); they are characterized by a sharp double valvular click, as if there were a double closure of the pulmonary valves. These sounds are of functional origin and may be disregarded as evidence of actual cardiac disease.

A study of apical adventitious sounds is of great practical importance because these are most apt to be confused with actual mitral disease. Most of the adventitious sounds in this area consist of soft blowing murmurs, accompanying, but not replacing, the first sound, and only slightly transmitted to the right and left of the apex. It has been asserted that these functional murmurs may even be transmitted posteriorly. I have not been able to corroborate this statement. These murmurs are occasionally loud; indeed, they may be as loud as some organic mitral murmurs. They usually vary with the position of the patient, and are sometimes capricious in that they are

present at some examinations and not at others. In addition to these adventitious apical murmurs are other abnormal sounds, scarcely mentioned in the literature, which I consider of great importance. I can best describe them as thrill-like or split first sounds. (See also Cardiac Neurosis, Chapter XVIII.) They are systolic in time and are usually confined to the apex. They are commonly found in tachycardias, or even with normal rates if the cardiac impulse is exceptionally vigorous. If the tachycardia disappears spontaneously, or if the heart rate or overaction can be quieted by having the applicant take and hold a deep breath, this thrill-like split first sound often disappears. Besides other characteristics distinguishing it from the typical *presystolic* rumble of true mitral stenosis (Chapter XIII), the adventitious sound described is not accompanied by the double click valvular sound so common in stenosis.

**4. Dyspnœa.**—The dyspnœa accompanying actual decompensation requires no comment here. I wish but to refer to the two types occasionally seen among the draft candidates. First, when found in tachycardia, the rapid heart action is the cause of the dyspnœa. The possible rejection of candidates should then be based upon the observation of the tachycardia along the lines already discussed, and not upon the dyspnœa. The second type of dyspnœa is that accompanying the *subjective* feeling of palpitation, with a perfectly normal, or even somewhat slow, heart rate. It is found more often in civil practice among neurotic individuals (Chapter XVIII) than among draft candidates, but is sufficiently frequent to deserve mention. It is not accompanied by any evidence of decompensation and is not appreciably increased even by sustained effort. It is a symptom which may, of course, be simulated by the candidate, but it is not a sign of cardiac disease and hence does not excuse the candidate from service.

## CHAPTER XXX

## THE ROLE OF THE HEART IN LIFE INSURANCE EXAMINATIONS

I believe that examination for life insurance is basically nothing more than the application of *objective* clinical medicine, for one cannot depend upon the history of the life insurance applicant.

The fact is fundamental and axiomatic, but will still bear repetition, that a person giving no history of infection, with a heart that presents perfectly normal sounds, with no murmurs, no enlargement, no overaction, no arrhythmia, no undue tachycardia, and associated with no dependent edema, no distress or dyspnœa after moderate exertion and no abnormal blood pressure reading, possesses a normal organ. In fact, it is more than normal; its an *ideal* heart, and from the standpoint of physical diagnosis and actual experience such an organ is indeed rare. This rarity alone forces us to consider as normal or satisfactory, from the clinical and insurance standpoints, hearts that are less than ideal, and to sift out a "standard" heart, despite murmurs and other apparent anomalies—I stress the word "apparent." Indeed, our increasing clinical experience, as the result especially of what was learned about the heart in the World War, has gradually, yet surely, weaned us away from the notion that the ideal is the only expression of an organically sound heart. And it behooves those who wish to advance and keep in touch with real progress in medicine—and this surely applies to the life insurance examiner—to take full cognizance and heed of facts as they are gleaned from actual clinical experience.

Since we rarely find an ideal heart such as outlined, let us see to what extent and in what direction deviations may occur, and yet have a heart that can be pronounced sound from the life insurance viewpoint.

During the routine study and examination by the life insurance examiner, certain outstanding queries should be constantly in his mind. First and foremost he must ever be on the lookout for any evidence of actual heart failure. With this presumably ruled out, he should be able to decipher, so to speak, cardiac abnormalities which may present themselves. He must ask himself whether such abnormalities, if present, are of functional or organic origin. Do they now or may they in the future interfere with the efficiency of the heart? Are murmurs present? If so, what is their time relationship to the cardiac cycle? Are they transmitted or not? Where are they best heard? Are cardiac irregularities present? If so, are they of functional or organic origin? It is, of course, understood that in one form or another, either in the medical blank or otherwise, many or all of these questions demand an answer from the examiner. Unless, however, there is a clear

mental background as to the bearing of these questions upon the efficiency of the heart and upon the probability of circulatory failure and, later, death from heart disease, they lack point and directness. Indeed, I have often thought it would be a wiser plan to give the examiner more scope, to perhaps discard or simplify the questionnaire, and to have the examiner briefly note abnormalities if present, and their bearing upon the soundness of the heart.

It may be here remarked that companies that do only a "standard business" should be more vitally interested in these questions than those who do also a "substandard business," for if some assumed "circulatory defects" have but an academic or scientific significance and are not indicative of heart disease or of a shortened span of life, such individuals should surely be accepted at normal rates, possibly after re-examination.

What constitutes heart failure from the life insurance standpoint and how can it be detected? The obvious signs are edema, dyspnœa, enlarged liver, pulmonary congestion; these are comparatively late developments. The only early sign, easily detected, is an unnatural, abnormal shortness of breath after comparatively brief exertion; this is the best, easily ascertainable and most tangible evidence we possess of decrease of the normal cardiac reserve power. In availability, simplicity and accuracy it surpasses all other functional tests. Abnormal breathlessness can be elicited by having the patient go through some simple exercises, such as rapid walking, hopping, bending, etc. Mere tachycardia after exercise, especially in younger individuals, even when lasting an abnormally long time, is not a sufficiently accurate index of loss of cardiac reserve, for tachycardia is sometimes long continued in perfectly normal hearts after exercise.

When individuals are secretive and do not wish to cooperate with the examiner, a fair index of the average daily cardiac power can be gauged by guarded questions as to the amount of usual exercises indulged in either at work or play.

What is a circulatory defect or abnormality? Cardiac failure, just described, is naturally the prime one. Insurance companies, however, usually emphasize cardiac irregularities and murmurs as other important ones. With reference to cardiac irregularities, the preponderance of those encountered, both in life insurance and clinical work, are sinus arrhythmia and extrasystoles. It is now generally held that sinus arrhythmia is a physiological phenomenon in the majority of cases. Its detection is easy without instrumental methods; there is alternate slowing and quickening of the heart, with the respective inspiratory and expiratory phases of respiration. When sinus arrhythmia accompanies organic disease, as it occasionally does, the evidence of the latter is unmistakable. With this exception applicants with sinus arrhythmia should be regarded as normal risks.

Extrasystoles (premature contractions, or dropped beats, as they are popularly called) are also easily detected in most instances. Since, in insurance examinations, the applicant may purposely withhold facts, his sensa-

tions are not dependable, and hence cannot be used as guides. The diagnosis is readily made by the fact that there is a weaker premature beat at the heart, accompanied or not by a weaker pulse beat, and usually followed by a somewhat long compensatory pause (Chapter XI). Is the extrasystole of functional or organic origin? The first part of the question—is it functional?—is in itself a concession toward a modern view in cardiology, only gradually gaining credence in life insurance work, for it was originally held that extrasystoles are always indicative of a muscular cardiac impairment. Those who have observed extrasystoles accompanying many types of extracardiac disturbance, for example, dyspepsia, are impressed with the view that they are often of functional origin, that they do not impair the efficiency of the circulation and that in themselves they do not indicate heart disease. Indeed, the insurance examination itself may produce extrasystoles in a nervous individual. Extrasystoles can, of course, also accompany organic cardio-vascular disease, hypertensive or otherwise. But here again the signs of disease are usually outspoken. Here, then a simple rule can be supplied for the examiners, namely: When extrasystoles (dropped beats) occur infrequently and are not accompanied by obvious cardiac disease, they should not be considered as evidence of impairment of the risk.

Constant rapid heart action, not truly an arrhythmia, requires brief mention. It is not always a sign of heart disease and is often and easily caused by nervousness incidental to the examination. The manner of the examiner frequently influences this. The following expedient to slow the heart is worth trying: The patient is asked to breathe deeply and to hold his breath a moment at the end of inspiration; this usually slows the nervous type of tachycardia. It should also be emphasized that rapid heart action, *per se*, can cause functional thrills and murmurs, to be mentioned later; these are usually systolic and are found at the apex. Hence, the importance of slowing the heart to exclude such functional adventitious murmurs and thrills. If it be impossible to slow the heart rate, a re-examination should be arranged for.

We may now consider murmurs and thrills. At the outset, I wish to emphasize this statement: That heart disease may be present without murmurs, and murmurs without heart disease. The importance and truth of this should be recognized. It comports well with what we know of the frequency and innocence of adventitious cardiac sounds—so-called functional murmurs. When these are definitely diagnosed as functional, it is proper to regard the applicants as standard risks. Up to the present, however, I believe many companies do not accept such a view in spite of the fact that re-examination often shows disappearance of these murmurs, and disappearance is almost positive evidence of their innocuousness. Although subject to many exceptions, functional murmurs are usually softer, less intense and are not transmitted to the same extent as organic murmurs. They are systolic in time in the great majority of instances. Rather than enter into the etiology of

functional murmurs, or into the characteristics of organic ones, it were better to briefly indicate simple methods of differentiation.

For insurance purposes, functional murmurs may be divided into those at the right base, at the left base and at the apex. They vary in intensity from faint to the rather harsh. Functional right base murmurs are systolic in time. They are differentiated from the organic aortic, stenotic or arteriosclerotic ones in this region by the fact that the latter produce a loud, harsh, vibrant systolic murmur, often transmitted to the carotids; there is sometimes in addition a rough palpable thrill.

Functional left base murmurs are, with very rare exceptions, systolic in time. They constitute the so-called cardio-respiratory murmurs. They are usually whiff-like and are confined to the left interspace. Occasionally they are blowing in character and are heard over the third interspace, and even further downward. They are ordinarily affected by respiration; during inspiration they become fainter. The cardio-respiratory murmur can readily be distinguished from the organic lesions (pulmonary stenosis and patent ductus arteriosis) at the left base, for these produce rough murmurs and thrills.

Murmurs at the apex, that is, over the mitral area, are more puzzling to differentiate as regards organic and functional. The history of rheumatism—an important guide in private practice—it may be impossible to learn from the applicant. Functional murmurs at the apex are occasionally as loud as the softer systolic ones sometimes heard in true mitral regurgitation. When evidence of heart failure accompanies the murmur the decision naturally is easy. But quiescent mitral regurgitant lesions often show no loss of cardiac reserve; there is no decompensation, the only physical sign is the murmur. Soft apical murmurs accompanying (not replacing) the first sound and but slightly transmitted are functional in the majority of instances. They are more apt to disappear with change of position of the individual, although I have not found that either this or increase of intensity of the murmur after exercise constitute sufficiently sharp diagnostic criteria between border-line functional and organic mitral murmurs. If, at another examination, the murmur is not heard at all, it argues very strongly in favor of an inorganic, functional murmur. Apical murmurs open to question should be re-examined. If finally regarded as probably functional, it seems to me they should be accepted on some plan.

Thrills over the base—right or left—are evidence of organic disease and are accompanied by auscultatory evidence of valvular damage.

In addition to these adventitious apical murmurs there are other abnormal sounds, scarcely mentioned in the literature, which I consider of great importance. I can best describe them as split, thrill-like first sounds, or even actual thrills (Chapters XVIII, XXIX). They are systolic in time and are usually confined to the apex. They are commonly found in tachycardia, or even with normal rates, if the cardiac impulse be exceptionally vigorous. If

the tachycardia disappears spontaneously, or if the heart rate or overaction can be quieted by having the applicant take and hold a deep breath, or by having him lay on his back, this thrill-like split first sound often disappears, a definite evidence of its innocent nature. Besides other characteristics distinguishing it from the typical presystolic rumble of true mitral stenosis, this adventitious sound is only exceptionally followed by the double-click valvular sound so common in stenosis. A further differentiation from mitral stenosis is the time relationship of the latter murmur. To repeat, the apical murmur is *systolic* in the functional cases and *presystolic* in the organic. Functional cases of the type I have described may be safely accepted as unimpaired risks.

The size of the heart as delimited by percussion is made much of in insurance examinations, but hypertrophy and enlargement are not as readily diagnosed as is popularly believed. Indeed, in the hearts just described, vigorous overaction alone frequently gives the impression of enlargement, an assumption which the X-ray often shows as fallacious. It may perhaps surprise life insurance examiners, when I state that I believe as time-honored a method as percussion has but a very limited sphere in the cardiac examination of the normal or of the assumed hypertrophied heart. Indeed, I believe inspection and palpation more valuable than percussion. The detailed reasons and an excellent clinical method for outlining the heart are given in a previous chapter (Chapter XIII).

There is much variability in the size and diameter of normal hearts (Chapter XII), they vary so much with the contour of the chest and the muscular makeup of the individual that wide differences in size may exist and yet the heart be normal. A heart with hypertrophy sufficiently pathological to exclude a risk will usually present other evidences of cardiac disease—hypertension, valvular murmurs, loss of cardiac reserve. All of this leads me to the important life insurance deduction I wish to make, namely, hearts *presumably* enlarged, but *in other respects normal*, might safely be accepted as normal risks.

I shall not enter here into the question of blood pressure (Chapter XXV). It is, however, of interest to note, since it has a bearing upon the subject of a "standard" circulation, that I do not consider low blood pressure (hypotension)—no matter how low—of any important significance from the standpoint of an organically sound heart. Such individuals often have many *symptoms*, chiefly giddiness, fatigue and a tendency to faint; in fact, they sometimes do faint. But the point I wish to emphasize is that the normal span of life does not seem to be interfered with, except possibly that a person falling in such a faint may become injured and even be killed. My own experience has been that low blood pressure is perfectly compatible with an organically sound, normal heart. I naturally leave out of account that type of low blood pressure which occasionally accompanies obvious severe cardiac decompensation. I also make an exception of those rare cases of coronary disease

occasionally found in middle life, in which hypotension may be the only *objective* evidence of myocarditis.

I have not entered into the advisability of accepting applicants with definite organic lesions. As a rule, this would apply to those with mitral regurgitant lesions quiescent and stabilized for several years, and without hypertrophy. Tangible evidence of a quiescent state may be gained from the fact that the applicant has regularly followed his usual occupation. The question of accepting such risks must rest upon the willingness and general policy of companies to insure, under sub-standard rating, individuals with stabilized lesions in whom the disease in no way affects the circulatory function.

I have attempted to portray some of the modern cardiological conceptions as applied to life insurance examinations in order to simplify the interpretation of some of the commoner circulatory abnormalities, and to indicate how decisions based upon modern clinical methods of examination may be arrived at by the usual clinical examination. All this, of course, presupposes good knowledge of cardiac physiology, as well as clinical experience. Some of my statements may perhaps seem questionable and not in accord with the insurance viewpoint. But general clinical knowledge should be in a sense a practical laboratory and have an influence on insurance statistics. Indeed, clinical experience in its broader aspects might well be a guide. The life insurance examiner should avail himself of newer cardiological viewpoints. Lessons taught by the X-ray, electrocardiograph and polygraph should be made practical and pertinent, and be intimately intertwined with the every-day work of the examiner.

## CHAPTER XXXI

## GRAPHIC METHODS—HEART SOUND REGISTRATION—PHONO-CARDIOGRAMS

Perhaps the crudest beginnings of the graphic representations and registration of heart sounds were the manometric flames of Koenig. The apparatus used was a thin-walled box one of whose sides consisted of thin membrane. The box was placed in circuit with illuminating gas by means of a tube connected with the gas main. The tube of exit was connected with the gas burner. The sound waves—for example, the human voice—were made to impinge upon the membrane. These compressed or rarified the gas contained in the box. The volumetric variations were transmitted to the burning flame with consequent variations in its height, corresponding to the voice sounds. By means of a mirror the image of these variations was reflected upon a photographic plate and thus registered. Another old instrument for registering sounds was Scott's phonautograph, invented in 1856, the forerunner of the modern phonograph. The phonograph itself has also been used for the study of heart sounds.

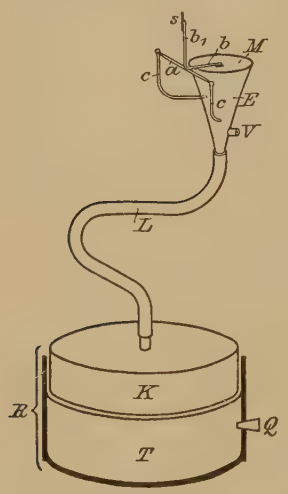


FIG. 285.—Ohm's gelatine membrane apparatus.
M, Gelatine membrane; A, lever; CC, carrier for the lever; S, small mirror; V, stop cock to have the system communicate with the open air. (*After H. Gerhartz.*)

Various other appliances, non-electrical in nature, have been devised for heart sound graphs. A few will be briefly described.

Ohm's Gelatine Membrane Apparatus (Fig. 285) consists of a tiny mirror glued to a gelatine membrane by means of a few fibers of absorbent cotton. This vibrating mirror system is in circuit with the source of the sound (the chest wall) by means of two cylinders: The one to be applied to the chest wall is closed off with a thin wooden membrane at its "chest" side, and a rubber membrane at the other. Upon this cylinder rests another closed by a rubber dam. The second cylinder contains a tube leading to the registration apparatus. The reason for two cylinders is dampening of the coarse sounds derived from the chest wall itself. The reflections of the tiny mirror resting upon the gelatine membrane are registered upon a photographic film.

In Gerhartz's apparatus (Fig. 286) a small reflector is kept steady by a magnet placed near it. The mirror is backed by a small thin iron sheet; on

either end of the mirror are two small perforations which carry fine needles that are connected respectively with the north and south poles of a magnet. The mirror is fastened to a membrane with a fine bamboo splinter glued to its center. The membrane itself consists of a collodion film 20 m.m. in diam-

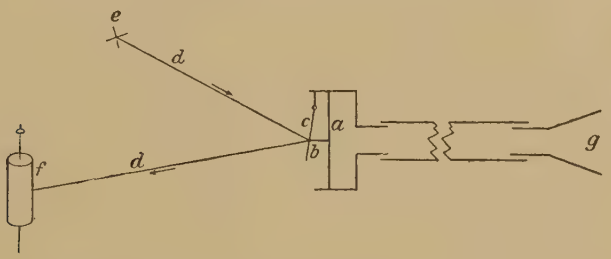


FIG. 286.—Schematic view of Gerhartz's heart sound registration apparatus. (*After Gerhartz.*)
A, Membrane; B, mirror support; C, mirror; D, light ray; E, lamp; F, film; G, sound funnel and tube.

eter. The phonendoscopic tube which carries the sound is only 6 m.m. in diameter, and so placed that the sound is conveyed to the center of the collodion film.

Several methods for the registration of heart sounds have been devised upon the basic principle of the interference rings of Sir Isaac Newton. They depend upon the following optical principles: If a sharply curved plano-convex lens is placed with its curved surface upon a flat glass plate, and a homogenous light is focussed upon the plate, optical interference rings will be produced which change with the change in distance between the two opposed glass

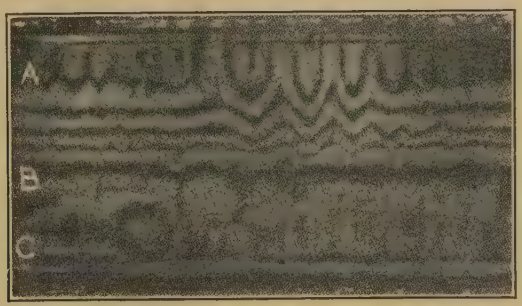


FIG. 287.—Photographic records of normal heart sounds (one cycle) based upon the principal of Newton's interference rings. (*After Austin.*)
A, normal heart sounds; B, apex tracing; C, time curve.

surfaces. Holowinski was the first to apply this principle to heart sounds. He used a specially constructed microphone to collect and magnify the sounds from the chest wall. The heart sounds then excited a telephone upon whose diaphragm there was adjusted an interference optical system such as just described. A modern adaptation of the Newtonian principle is found in the

micrograph of Crehore. The optical interference instrument here consists of a metal tambour, the upper surface of which is a thin brass diaphragm. To the center of the latter a small mirror is attached; immediately above the mirror is a stationary glass lens. When used for transcribing cardiac pulsations, a cup connected by rubber tubing to the interference instrument is placed over the cardiac apex. The movements in the enclosed column of air cause the telephone diaphragm, and with it, the mirror, to vibrate. When the rays of a mercury vapor lamp are thrown upon the optical system, the change in distance between the glass surfaces causes movements of the light interference rings. The rings are then photographed by a kymophotographic apparatus. In order to adapt the Crehore instrument to the registration of

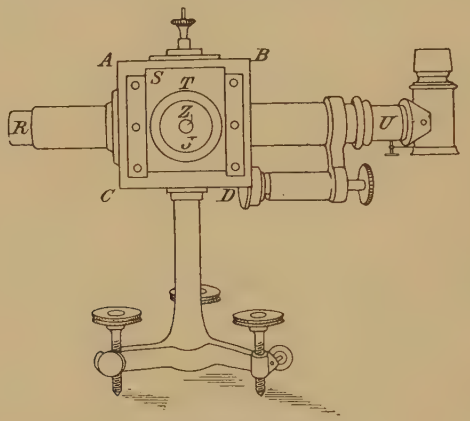


FIG. 288.—Weiss phonoscope.
ABCD, Box; S, slide; T, tube; T, plate; Z, opening, 1 c.m. in diameter, over which the soap film is spread. (*After Gerhartz.*)

sound records and to make it more sensitive, a rubber diaphragm was substituted for the metal one. Figure 287 shows the registration of a complete cardiac cycle. In addition to the sound records, a simultaneous apex tracing had been taken with another interference micrograph.

The Weiss soap-film phonoscope (Fig. 288) consists of a metal box with a side opening into which fits a centrally fenestrated plate. The latter is covered by a thin soap film. On the top of the box rests a fine silvered rectangular glass lever connected with the soap film by means of an adjustment screw. The sound is transmitted from the chest wall to the soap film through the medium of a suspended funnel; the resultant vibration produces movements in the glass lever. These movements are illuminated and magnified by means of a microscope and a light projection system focussed upon the lever; a second lens projects the image upon the photokymograph.

The Frank segment capsule (Chapter VII) has also been employed in the registration of heart sounds. The sound transmitting apparatus consists of a stethescope bell or phonendoscope which is connected to the recording capsule

by means of a rubber tube having an adjustable lateral opening. When the lateral opening is closed, heart sound vibrations are obtained, some of which are superimposed upon the apex tracing (Fig. 289). In order to exclude the coarser apical vibrations, the lateral opening should communicate with the outer air.

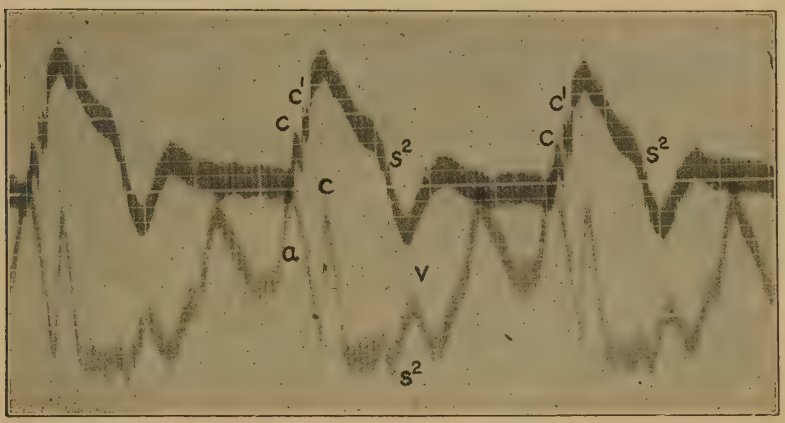


FIG. 289.—Heart sounds, apex tracing (above) and supraclavicular venous pulse (below), taken with the Frank segment capsule. (*After Wiggers.*)
*acv*, Venous pulsations; Apex (upper) tracing; C, initial vibration; $C^1$, valve vibrations; $S^2$, vibration due to second sound (transmitted to apex and jugular tracings).

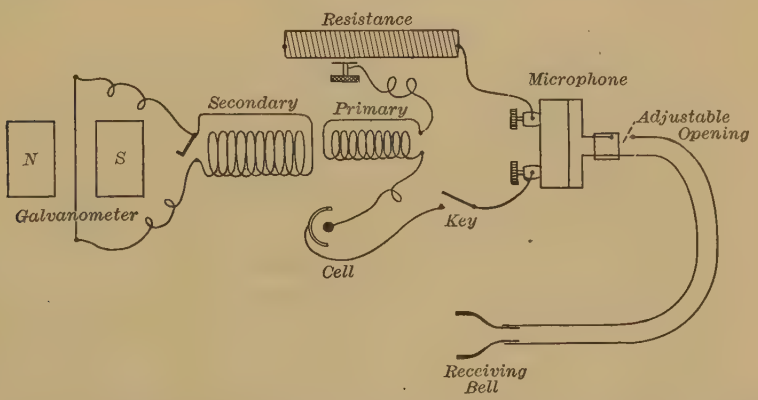


FIG. 290.—Scheme of microphone apparatus and connections with the electrocardiographic apparatus for the taking of phonocardiograms. (*After Wiggers.*)

**The Phonocardiogram.**—Of all methods of taking graphic sound records, that in which the electrocardiographic apparatus is employed is the most accurate. While there are some differences in the type of the sound-receiving apparatus, they all consist essentially of a receiving bell to be applied to the chest wall. The bell is connected to a microphone by means of a rubber tube; the rubber tube has an adjustable opening through which it may be made

to communicate with the outer air. The microphone is placed in circuit with the string galvanometer by means of a coil and rheostat. Figure 290 shows a compact apparatus for the taking of phonocardiograms.

For the actual registration of the sounds, it is necessary to have the string at about four times the tension employed in taking an electrocardiogram;

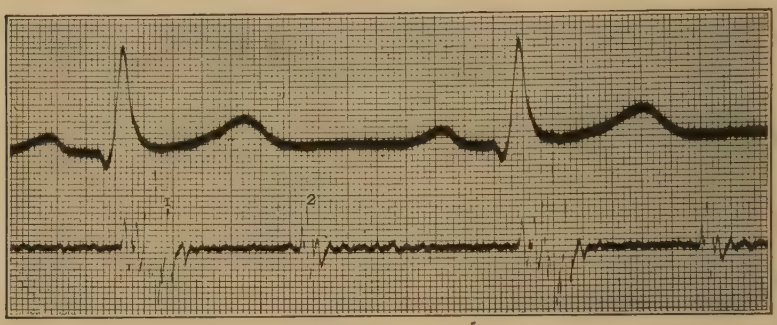


FIG. 291.—Normal phonocardiogram with simultaneous electrocardiogram (upper tracing); phonocardiogram (lower tracing). (*After H. B. Williams.*)
1. First sound; 2, second sound.

in other words, instead of one centimeter deflection for 1 millivolt of current (Chapter VIII), the string deflection should be 1 centimeter for 4 millivolts of current when phonocardiograms are to be taken. The heights and other characteristics of phonocardiograms cannot be standardized because it may

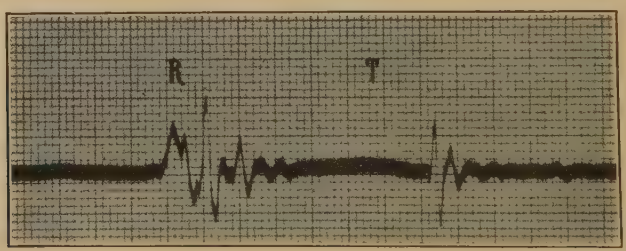


FIG. 292.—Phono- and electrocardiogram taken simultaneously with one string. (*After Fahr.*)

be necessary to change the string tension in order to record various abnormal sounds and murmurs.

A normal phonocardiogram is shown in Fig. 291. Two strings have been used, one for the electrocardiogram, the other for the sound record. In this manner, it becomes easy to measure not only the duration of the sounds, but also to see and measure their incidence and relation to the events in the cardiac cycle (Fig. 57), for, with our knowledge of the electrocardiogram, we are enabled to fix the relation of cardiac sounds and murmurs in systole and diastole, as well as in the cardiac pressure curve (Fig. 57).

Although it is necessary to employ two strings for purposes of accurate study of the time relationships of the sounds with cardiac events, one string alone may also be used. The patient is then connected with the galvanometer as for an ordinary electrocardiogram, the phonocardiographic apparatus is put into circuit at the same time. In this manner the heart sound records

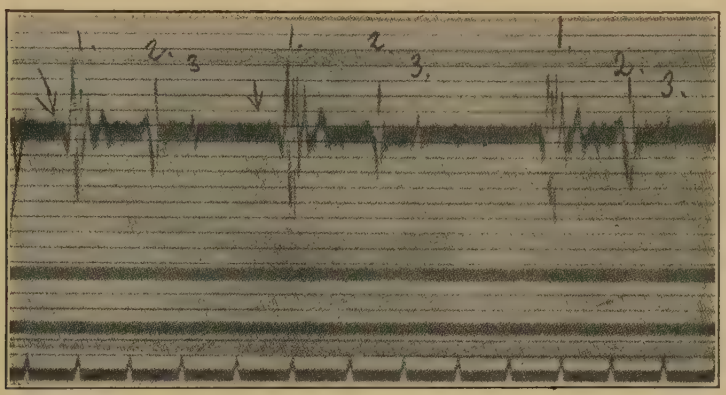

FIG. 293.—Phonocardiogram of third heart sound. (*After Bridgman.*)

will produce vibrations which will break the symmetry of the electrocardiogram, but enough of the landmarks of the latter are usually visible to fix the auricular and ventricular events, and thus to fix the time relationship of the phonocardiographic events as well (Fig. 292).

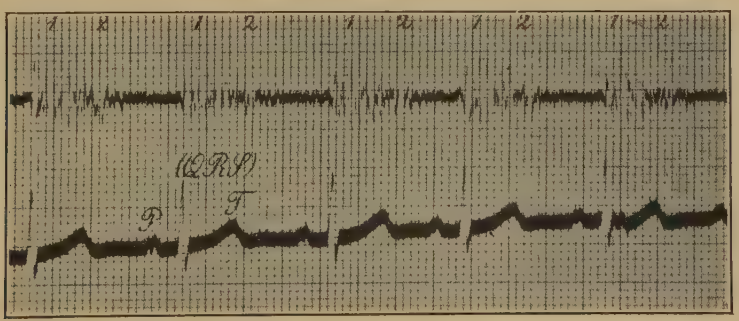

FIG. 294.—Phonocardiogram taken from apex. The systolic murmur (1) occupies the entire systolic period. (*After Battaerd.*)

In their appropriate connections (Chapter XIII), the causes of the normal sounds, and the etiological, diagnostic and clinical significance of abnormal sounds and murmurs are fully dealt with. Here it is sufficient to reproduce the more important types of normal and abnormal phonocardiograms, and to indicate how this method of sound registration is an aid to auscultation.

Reference to the simultaneous phono- and electrocardiogram (Fig. 291, 292) shows that the beginning of the first sound takes place very shortly after the onset of the R wave. Since it is practically certain that the R wave occurs almost simultaneously with ventricular contraction, it seems probable that at least the onset of ventricular contraction causes no sound. This has an important clinical application in the assumption that weak heart sounds are indicative of weakened or diseased heart muscle (Chapter XIII).

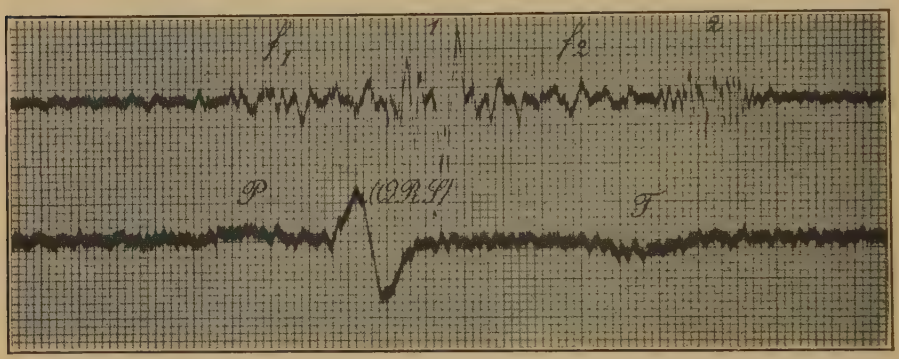


FIG. 295.—Phonocardiogram showing a diastolic (f 1) and a systolic (f 2) murmur. The photographic plate is traveling at a fast rate (100 m.m. per s.) in order to get a detailed curve. (*After Battaerd.*)

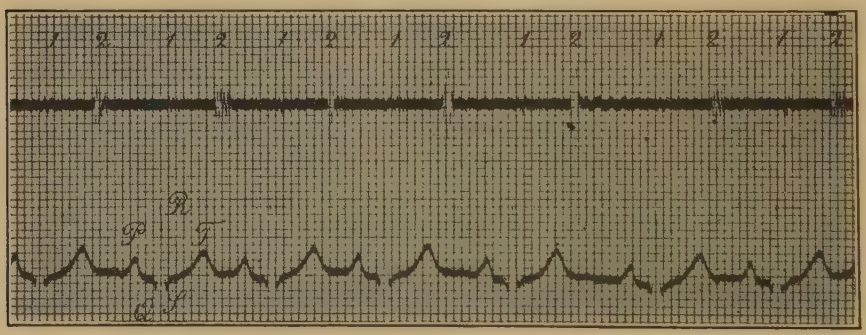


FIG. 296.—Phonocardiogram showing an accentuated second pulmonic sound. The microphone was placed over the pulmonary valve. (*After Battaerd.*)

In addition to the phonocardiograms of the first and second sounds, occasionally a phonocardiogram of a third heart sound may be obtained (Fig. 293). Types of systolic and diastolic murmurs are shown in Figs. 294, 295. The phonocardiogram of an accentuated second sound is shown in Fig. 296. The figures and accompanying legends sufficiently describe the time relationships and types of the murmurs.

Phonocardiograms are undoubtedly of great aid in helping to solve some of the physiological factors connected with sound production. This is accom-

plished by a comparative study of their time relationship with the electrocardiogram, with intraventricular pressure curves and with the time of valve closure (Fig. 57). In this manner, graphic sound records are of value, particularly in determining the question of the muscular element in the production of heart sounds.

The special clinical value of phonocardiograms rests in the graphic representation of murmurs, for phonocardiograms may give added information regarding the time-relationship and intensity of adventitious sounds. It should be stated that the phonocardiogram gives no information regarding the source of a murmur; it simply gives, as stated, its time relationship and relative intensity. And while it will always transcribe the murmurs, it has this disadvantage as compared with auscultation in that the trained ear invariably selects the sound or murmur which seems of greatest importance clinically, and follows it through the heart cycle (Chapter XIII). Besides, faint murmurs do not always register in the phonocardiogram.

I believe the great clinical advantage of the phonocardiogram is its ability to transcribe the time relationship of a given murmur, which upon auscultation may be open to question as to whether it is systolic or diastolic. To clear up such a problem is naturally of great diagnostic importance, especially when dealing with the probability of the two valvular lesions: Aortic regurgitation and mitral stenosis. As an instance, I recall a case of aortic aneurism, visibly pulsating in the jugulum, that required a phonocardiogram in order to determine whether a murmur which was quite loud on auscultation was systolic or diastolic in time. The phonocardiogram showed it to be diastolic. Even here, however, careful palpation over the aneurism at the time of auscultation would have given the desired information; for if the murmur were not synchronous with the pulsation of the aneurism (and therefore with cardiac systole), it must of necessity be diastolic.

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## CHAPTER XXXII

## CLINICAL SIGNIFICANCE OF THE ABNORMALLY WIDE VENTRICULAR DEVIATION IN THE ELECTROCARDIOGRAM

Because of the importance and clinical significance of myocardial disease, attempts are constantly being made to interpret the condition of the myocardium from the electrocardiogram.

In an electrocardiographic study of a series of patients with valvular and myocardial disease, reference to which has already been made (Chapter IX), I observed a characteristic which seems of importance in the diagnosis of myocarditis, namely, the abnormal width of the main deviation of the ventricular complex, commonly called the R-wave or the first ventricular spike. The width of the normal R-wave has been variously estimated. Hirshfelder estimates it as 0.02 of a second; Lewis as from 0.03 to 0.04 of a second. Though not directly mentioned by Einthoven or Nicolai, measurements of a number of their normal ventricular complexes give an average width of less than 0.05 second. All the measurements have reference to R waves of normal form, that is, to those that are neither split or splintered, and whose sides form rectilinder lines which meet at a sharp angle and form a sharp apex. In the measurements of my cases I have included only clean-cut deviations of normal form. As an arbitrary standard in my series I have adopted a base line width of 0.07 or over as representing abnormally wide deviations. From my own measurements of many ventricular deviations of patients with normal hearts, and from the normal standards of others, I believe that 0.07 of a second (or over) probably represents a deviation which has taken an abnormally long time for its completion. Indeed, in only one instance (M.G., Case 13) have I found the R wave of a normal heart wider than 0.07 of a second.

Robinson pointed out the clinical importance of abnormally wide and splintered ventricular complexes as evidence of marked functional changes in the heart (Chapter IX). Oppenheimer and Rothschild have shown that some cases of myocardial disease involving the subendocardial myocardium (intraventricular block) present an R complex of low voltage, wide base and irregular sides (Chapter IX). They believe this occurs because myocarditis in this location interferes with the rapidity and uniformity of the spread of the electrical impulse through the heart by involving the superficial end branches of the conduction system. These observers have confirmed the deductions drawn from the electrocardiograms by the pathologic study of a number of necropsies. Eppinger and Rothberger induced experimental destruction of various parts of the cardiac musculature by freezing with ethyl chlorid, or

by the injection of destructive substances. Electrocardiograms were taken before and after the injections. When, for example, a strong nitrate of silver solution was injected in the deeper layers of the left ventricle either at the base or apex, there was a marked change in the ventricular deviation. Within five minutes after the injection the descending limb became shorter so that it no longer touched the base line; this shortening became progressive so that in about twenty minutes the R and T deviations formed practically a monophasic curve. Injections of nitrate of silver in the superficial layers, or in the anterior and posterior papillary muscles were not followed by the foregoing changes in the electrocardiogram. Injections in the right ventricle were not regularly followed by electrocardiographic changes; the most typical consisted in a widening and prominence of the S and T deviations. I mention these experiments in some detail because they have an important bearing on the present clinical study. We must assume that we clinically meet with focal myocardial changes probably at times similar in location and destructiveness to the experimental lesions quoted; yet in only a single instance in a study of very many cases of marked cardio-sclerosis did I observe an electrocardiogram which resembled that derived from Eppinger and Rothberger's experimental injection in the deeper layers of the left ventricle. It therefore seems unwarranted to draw deductions of the state of the pathologic process in the human heart from the type of experimental damage already quoted. This discrepancy may rest on the fact that *rapid* experimental destruction of the myocardium produces some special change of the electrocardiogram, or that in almost every instance in the human heart where there are marked localized changes, there is also damage in other portions of the myocardium; that is, the local damage is only part of general myocardial disease.

In a description of cases assuming defective conduction in one of the main branches of the auriculo-ventricular bundle (Chapters IX, X), Carter describes as characteristics of these lesions, a Q-R-S time exceeding 0.10 second, increased amplitude of the R deflections, and T waves, often exaggerated and usually in a direction opposite to that of the prominent initial deflection. The initial deflections themselves are very frequently atypical in form in at least one lead; bizarre forms are common. The assumption of branch bundle lesions in these cases has recently been questioned (Oppenheimer and Rothschild).

In the cases that I report there has been no reason to assume an abnormal path for the impulse, for the electrocardiograms possess none of the characteristics of the aforementioned groups. Except for the abnormally wide base line, the electrocardiograms are of the usual normal types in form and size. Some of the deviations are tall. That amplitude in itself, however, need not necessarily imply an abnormally wide base is shown in cases in which R I is quite tall though of normal width (Chapter IX).

A glance at the summary in the accompanying table of cases shows that, grouping them by their clinical characteristics, in all instances but two

(Cases 3 and 5), there was grave disease of the myocardium; that is, myocarditis represented an important part of the clinical entity. The two exceptions were cases of rheumatic aortic disease in both of which ventricular hypertrophy was extreme. That abnormal prolongation of the time of the electrical impulse is not due to decompensation itself is evidenced by the fact, as noted in the clinical histories, that many of the electrocardiograms were taken when the patients were perfectly compensated. Ventricular dilatation *per se* as a cause for the abnormally wide R could be excluded. First, there was no evidence of any extreme dilatation, certainly not in the compensated cases. Secondly, in a study of a large series of cases of decompensated valvular lesions, in which cardiac dilatation was presumably a prominent feature, I found no abnormally wide R. Nor had I there found any correlation between hypertrophies—even extreme—and the width of the R deviation. Whether in the present study Cases 3 and 4 may be regarded as evidence of myocarditis in addition to hypertrophy, it is impossible to say.

Summary of Cases

| Case No. | Clinical diagnosis | Rhythm | Width of R in hundredths of second | Remarks |
|---|---|---|---|---|
| 1 | Myocarditis; coronary disease | Normal | R II = 0.07<br>R III = 0.08 | Arrhythmia and death |
| 2 | Luetic aortitis; myocarditis | Interpolated extrasystole | R I = 0.12<br>R II = 0.07<br>R III = 0.13 | |
| 3 | Rheumatic aortitis | Auricular fibrillation | R II = 0.07 | Massive ventricular hypertrophy; death in one year |
| 4 | Cardio-nephritis | Normal | R I = 0.08<br>R II = 0.08 | Precordial pains |
| 5 | Rheumatic aortitis | Normal | R I = 0.10<br>R II = 0.09<br>R III = 0.08 | Massive ventricular hypertrophy |
| 6 | Cardio-sclerosis | Normal | R I = 0.10<br>R III = 0.07 | |
| 7 | Cardio-nephritis | Auricular fibrillation | R I = 0.08 | Died in uremic coma |
| 8 | Myocarditis; arteriosclerosis | Sinus arrhythmia | R I = 0.08<br>R III = 0.08 | |
| 9 | Aortic aneurism | Normal | R II = 0.07 | Moderate amount of myocarditis at necropsy |
| 10 | Cardio-sclerosis | Auricular fibrillation | R II = 0.08<br>R III = 0.09 | |
| 11 | Cardio-sclerosis | Normal | R I = 0.12<br>R II = 0.08 | Aneurismal dilatation of the aortic arch |

It will be noted from the table that the electrocardiograms showed an abnormally wide R in one or two leads, rarely in three. In view of the fact

that the leads draw off the current from various cardiac areas—breadthwise in Lead I, diagonally in Lead II, and lengthwise in Lead III—one may possibly assume that the diseased myocardium lay more in one plane of the current than the other, thus interfering with the quick distribution and spread of the electrical current in that particular plane.

It remains to indicate this important fact that while the cases mentioned show abnormally wide ventricular deviations and are apparently indicative of severe myocardial disease, I have observed cases clinically similar to the above with perfectly normal electrocardiograms. The reason for this is not at present apparent.

The conclusions are given in another chapter (Chapter IX).

## REPORT OF CASES

Case 1.—A. G., man, aged 54, had complained for two years of indefinite precordial pains radiating to the left hand, and of attacks of dizziness. He had been unable to work for several months because of these attacks. On examination, the systolic blood pressure was 166, the diastolic, 88. The first aortic sound was blurred, the second somewhat accentuated. The other heart sounds were normal. There was slight edema of the legs. The urine contained no albumin or casts. Fluoroscopic examination showed a normal sized aorta; the ventricular outline was somewhat enlarged to the left. The electrocardiogram at that time showed normal rhythm; R II and R III were respectively 0.07 second and 0.08 second in width. The subsequent history of the patient was of interest. Following the use of digitalis and of theobromin sodium salicylate, there was gradual improvement in the symptoms for about one year. The patient then developed sudden attacks of tachycardia, each of which lasted several days. The symptoms during these attacks were mainly subjective and consisted in the uncomfortable recognition of the rapid heart action; dyspnœa was slight. About one week after the last attack he suddenly complained of precordial distress, followed by faintness. The patient was moderately dyspnœic. Auricular fibrillation with rapid ventricular activity was present. A diagnosis of coronary thrombosis was made. Five days after the onset of fibrillation, while sitting quietly on a chair and apparently quite comfortable, he suddenly gasped and died within a few minutes. The clinical diagnosis was myocarditis and coronary sclerosis.

Case 2.—W. Z., man, aged 57, entered the hospital with general anasarca and the usual symptoms of cardiac decompensation. This had been the third break of compensation within recent years. The physical signs revealed the presence of an aortitis and of left ventricular hypertrophy. This was corroborated by roentgenography, which showed in addition, aneurismal dilatation of the entire thoracic aorta, and ventricular enlargement which practically filled the lower left half of the chest. The Wassermann blood

examination was 4 plus. On antisyphilitic treatment, Karrell diet, digitalis and theobromin sodium salicylate, the patient quickly improved. The electrocardiogram taken when the patient was compensated, showed abnormally wide ventricular deviations in all leads, especially in the first and third. The R widths were respectively 0.12 and 0.13 second; R II was 0.07. The clinical diagnosis was syphilitic aortitis, myocarditis and left ventricular hypertrophy.

Case 3.—H. Y., male, aged 31, had acute articular rheumatism six years prior to admission. Six weeks ago he suffered from what at first appeared to be grippe; this was soon followed by fever and joint pains. He had never had any cardiac symptoms until recently. Examination revealed markedly throbbing carotid, subclavian, brachial and radial arteries. A rough systolic thrill was heard over the carotid and over the aorta in the jugulum. There was a loud, rough, double murmur over the right base. The liver was somewhat enlarged. The urine contained a trace of albumin and a few granular casts. The Wassermann blood examination was negative. The orthodiascope revealed an enlarged and hyperacting aorta, the left ventricle was moderately enlarged. The electrocardiogram showed auricular fibrillation; the width of R II was 0.07. The patient developed hemorrhagic erythema of the legs, and later, rheumatic joint manifestations. He died within three months. The clinical diagnosis was a double aortic lesion and moderate left ventricular hypertrophy.

Case 4.—I. K., woman, aged 57, complained for several years of a feeling of oppression in the chest and of numbness in the fingers of both hands. There were also occasional attacks of nocturnal dyspnea accompanied by precordial pains. The systolic blood pressure ranged between 180 and 220, the diastolic between 90 and 110. There was a metallic second sound over the aorta, and a systolic murmur at the apex. With the fluoroscope the aortic arch was found enlarged, the left ventricle moderately hypertrophied. The urine showed a trace of albumin and a few casts. R I and R III were each 0.08 second in width. The clinical diagnosis was cardio-nephritis.

Case 5.— E. O., woman, aged 21, had "growing pains" as a child. Her "heart trouble" began at the age of 10. The chief complaint was palpitation, especially after undue excitement. The systolic blood pressure was 180, the diastolic 30 m.m. The classical signs of a double aortic lesion and left ventricular hypertrophy were present. The orthodiascope revealed a violently pulsating aorta and massive ventricular hypertrophy, as well as enlargement of the cardiac shadow to the right. The electrocardiogram showed the widths of the R deviations as follows: R I = 0.09 and R II = 0.08.

Case 6.—A. K., physician, aged 53, had scarlet fever at the age of 15. Since then he had had occasional traces of albumin and a few casts in his urine. Of late, he has become somewhat dyspneic. The systolic blood pressure ranged between 175 and 190, the diastolic between 110 and 85. The second aortic sound was somewhat accentuated; there was a systolic murmur at the apex. The liver was somewhat enlarged. The urine contained

a trace of albumin, and granular and hyaline casts. There was slight edema of the legs. The blood Wassermann was negative. The orthodiascopic examination revealed a somewhat enlarged aorta and cardiac shadow. In the electrocardiogram, R I and R III were each 0.07 second wide. The clinical diagnosis was cardio-nephritis.

Case 7.—I. W., woman, aged 45, was brought to the hospital in a comatose condition. The only history obtainable referable to her present condition was that six years previously, when pregnant, she lost her eyesight (uremia?); labor was induced and her eyesight returned. At the present examination, her heart was found hypertrophied, there was marked accentuation of the second sound at the apex and base. The systolic blood pressure was 270 m.m. A blood culture was sterile. The electrocardiogram showed that R I was 0.08. Two days after admission the patient died in uremic coma. The clinical diagnosis was cardio-nephritis.

Case 8.—S. M., male, aged 53. He complained for several years of dyspnea and precordial pains, particularly on slight exertion. He had had transient attacks of Cheyne-Stokes breathing; at such times his pulse was arrhythmic, electrocardiographic curves showed that the arrhythmia was of sinus origin. The apex was in the sixth left interspace, 14 c.m. from the midsternal line. The heart sounds were faint, there was a soft systolic murmur at the apex and a slightly accentuated second sound at the base. The radial arteries were thickened and tortuous. The systolic blood pressure was 190 m.m., the diastolic, 110 m.m. The electrocardiogram showed that R I and R III were each 0.07 second wide. The clinical diagnosis was myocarditis and cardio-sclerosis.

Case 9.—J. J., aged 53, man, had a protuberant aneurismal tumor the size of a large orange, situated in the upper right side of the chest. There were loud systolic and diastolic thrills and pulsations over the aneurism, there was also a systolic murmur and a systolic thrust at the cardiac apex. The apex was in the sixth interspace, one inch outside the nipple. Though the patient was somewhat dyspneic at first, there was no marked decompensation. R I and R III were each 0.07. The aneurism was wired. After some weeks, there were frequent external hemorrhages from the aneurismal sac; the patient gradually succumbed. At necropsy, besides the aneurism, the ventricles were moderately hypertrophied and showed a moderate amount of myocarditis.

Case 10.—J. C., man, aged 60, was addicted to alcohol. During the previous two years, he had had several mild attacks of dyspnea and of edema of the legs; the last attack occurred one week prior to admission. The cardiac apex was in the fifth interspace, 12 c.m. from the midsternal line. There was a blowing systolic murmur at the apex, the second sound at the base was impure and slightly accentuated. The arteries were thickened. The electrocardiogram showed auricular fibrillation. R II was 0.08 second, R III 0.09 second wide. The clinical diagnosis was cardio-sclerosis.

CASE 11.—M. M., aged 65, had rheumatism several years prior to admission. She had been somewhat dyspneic for several years; of late this symptom has become more marked. The first examination revealed mucous rales over the entire chest. There was a loud systolic murmur heard over the upper right chest, transmitted into the carotids and to the left of the scapula posteriorly. There was also a loud systolic murmur over the mitral area. The blood pressure was normal. There was slight edema of the legs. The urine showed a slight trace of albumin and a few casts. In the fluoroscope, there was found aneurismal dilatation of the first portion and arch of the aorta; the heart was moderately enlarged. R I was 0.12 and R III 0.08 in width. The clinical diagnosis was cardio-sclerosis.

## CHAPTER XXXIII

## MATHEMATICAL CONSIDERATIONS UNDERLYING THE ELECTROCARDIOGRAM

To those interested in understanding some of the mathematical principles upon which the electrocardiogram is based, this chapter while somewhat obstruse, may be of importance.

In previous chapters (Chapters VIII, IX) the fact was emphasized that the electrocardiographic deviation represented the resultant of the differences of electrical potential existing at any given moment. In the case of the ventricles, it is in fact the resultant of separate activities of the ventricular chambers. The diagnostic significance of the direction of the R wave was also discussed. The elementary mathematical principles upon which differences in the R deviations depend now require brief comment.

Stress has been laid upon the fact that the size of the R peaks depends upon the angle formed by the line across the heart (represented by the "leads," Chapter VIII) and the "electrical axes;" the nearer the electrical axes approach parallelism to the leads, the taller the corresponding peaks; the nearer these approach the right angle, the smaller the peaks. The size of the deviation thus varies with the angle made by the lead and the electrical axes. In other words, the electrocardiogram is modified in each lead by the relation of the direction of the active current in the heart to that of the leads placed upon the extremities. Conversely, the angle made by these two lines may be computed from the differences in size of the deviation in the three leads.

**Manifest Size.**—Einthoven has differentiated the "actual" size of the deviations, as reproduced in the electrocardiogram, from their "manifest" size or the "manifest" difference of the electric potential. He defined the latter as that dimension in millivolts which is derived when the electrical axis and leads coincide: It is the maximal size of the electrical potential of the heart in the lead. The manifest size of any deviation is computed from the registered actual electrocardiogram. Einthoven and his pupils, Fahr and de Waart, have laid down mathematical principles by which it is possible to estimate the angles formed by the electrical axes with the leads. If, for purposes of simplification, the lines of the three leads be conceived as forming the sides of an equilateral triangle (Fig. 297 R. L. F.), and its middle point, H, represent the heart, the size of the R deviations in the three leads is obtained by the right angled projection of the deviations upon the electrical axis pass-

ing through the point H. From these, data are derived for the determination of the angle formed by the electrical axis and the leads.[1]

**Measurements of the Angles Made by the Leads and the Electrical Axis.** If smaller angles be discarded, and for purposes of clinical approximation, only those be considered which are multiples of 30 deg., the computation of E1:E2: (Fig. 297) is much simplified; for example,

When

$\alpha = 0°$, then $E_1 : E_2 : E_3 = 1 : +.5 : -.5$

$\alpha = 30°$, then $E_1 : E_2 : E_3 = 1 : 1 : 0$

$\alpha = 60°$, then $E_1 : E_2 : E_3 = +.5 : 1 : -.5$

$\alpha = 90°$, then $E_1 : E_2 : E_3 = 0 : 1 : 1$

etc.

From these numerical proportions, the angle $\alpha$ can be approximated within 30 deg. The "manifest" equals the actual size of R1 (E1, Fig. 297) when $\alpha$ equals 0 deg.; of R2 (E2) when $\alpha$ equals 60 deg., and of R3 (E3) when it equals 120 deg. For the exact determination of this angle, tables are required. In the Appendix to this chapter (q. v.) there is a table for approximation within 10 deg.; by the use of the second table (Interpolation Table) the exact angle may be computed. For example, if R1 equals 3.2, R2 equals 12.5, and R3 equals 9.3, then E1 (R1): E2 (R2): E3 (R3) = 3.2:12.5:9.3. To derive an approximation within 10 deg. (Table 1), 12.5, the tallest deviation, becomes the denominator, thus: $\frac{10}{12.5}$ 12.5. Substituting this fraction for 12.5 in the equation, we find E1: E2: E3 = 2.6: 10:7.4. Furthermore, from the same table we note these figures determine that the angle $\alpha$ falls between 70 and 80 deg.; and that E1 between 70 and 80 deg. has a value between 3.5 and 1.8, a difference of 2.7. In Table II (Interpolation Table, Third Column) the nearest approximation to this difference is 2.6, which is equivalent to 6 deg.; hence, $\alpha$ = 70 deg. + 6 deg. = 76 deg.

As another example, suppose T1 = 4, T2 = 1.5, and T3 = 3.5, we then have the proportion E1: E2: E3 = 4:1.5: − 2.5. To derive approximation within 10 deg. we multiply by the fraction $1\frac{0}{4}$, 4 being the tallest deviation. Then E1: E2: E3 = 10: 3.75: − 6.25. From Table I, this proportion shows that the angle $\alpha$ is between 0 deg. and − 10 deg., and also that E2 varies from 5 to 3.5. In Table II, its nearest approximation is 3.8, an angle of 8 deg. Hence $\alpha$ = 0 deg. −8 deg. = −8 deg.

[1] To insure mathematical accuracy it is necessary to measure the R deviations at identical phases of the heart cycle. With this in view, two galvanometers may be simultaneously employed; one to record sound records, the other to record the electrocardiograms. Although this method is necessary for absolute accuracy, measurements derived from the ordinary electrocardiogram are sufficiently exact for clinical purposes unless phasic differences of the heart cycle produce marked changes in the electrocardiogram.

Since the value of the angle alpha can be computed, it is possible by trigonometry[1] to determine the manifest size (Fig. 297) of the various deviations.

In a schematic diagram by Pardée (Fig. 298), based upon the Einthoven conception of the leads forming a triangle with the heart in the center, the angle $\alpha$ can be roughly measured by conceiving the direction of the electro-

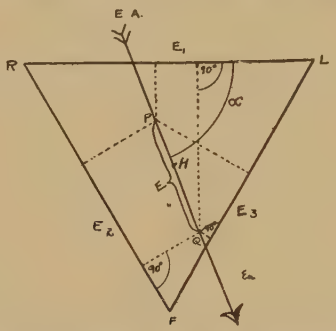


FIG. 297.—The right and left arms and left foot (designated R, L, F, respectively) form the angles of an equilateral triangle. The point H is in the center and represents the heart. If the arrow EA represent any given electrical axis, the angle it forms with the first lead (RL) be represented by X, and any given length PQ be called E, the right angled projection of its length will give its corresponding value in the various leads; that is, in L I it will equal E I; and L II, E2 and in L III, E3. The distance E1, E2, E3 are proportional, that is, E1 : E2 : E3. Since any angle of an equilateral triangle is equal to 60 deg., the following trigonometrical formulæ are derived:

$$E1 = E \cos \alpha;$$
$$E2 = E \cos (\alpha - 60°);$$
$$E3 = E \cos (120 - \alpha);$$
$$E3 = E2 - E1.$$

The formula E3 = E2 − E1 is of special importance, for, given the heights in deviations in any two leads, the height of the remaining peak may be derived. (*After Einthoven, Fahr and De Waart.*)

cardiographic current as lying in one of six sectors of 60 deg. each. Thus, currents or electrical axes between + 30 deg. and + 90 deg. (the normal segment) give + R1 + R2 + R3.

Axes between + 90 deg. and + 150 deg. give − R1 + R2 + R3
Axes between + 150 deg. and − 150 deg. give − R1 − R2 + R3
Axes between − 150 deg. and − 90 deg. give − R1 − R2 − R3
Axes between − 90 deg. and − 30 deg. give + R1 − R2 − R3
Axes between − 30 deg. and + 30 deg. give + R1 + R2 − R3

These results are diagrammatically shown in Fig. 299.

[1] The formulæ by which the value of E (the manifest size) may be derived are:

$$E = \frac{E1}{\cos \alpha}$$

$$E = \frac{E2}{\cos (\alpha - 60°}$$

$$E = \frac{E3}{\cos (120° - \alpha}$$

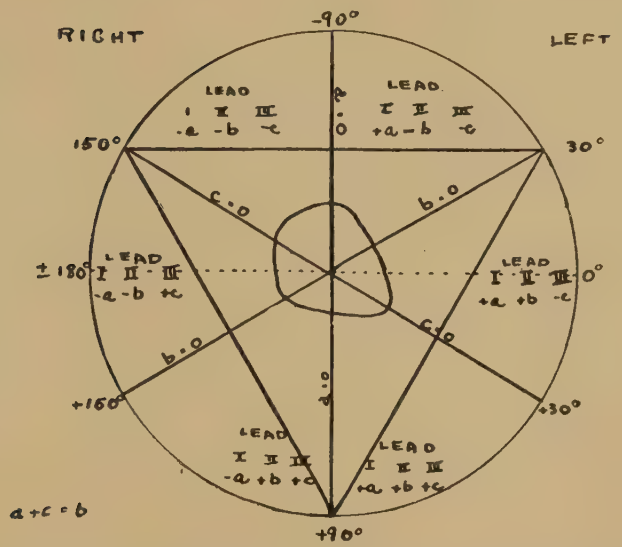


FIG. 298.—a, b, c, represent the numerical value of the deflection caused by an action current in leads I, II, III, respectively. b = a + c and will be directed upwards or downwards according as a and c are directed; + before the letter signifies an upward deflection; − signifies a downward deflection. (*After H. E. Pardee.*)

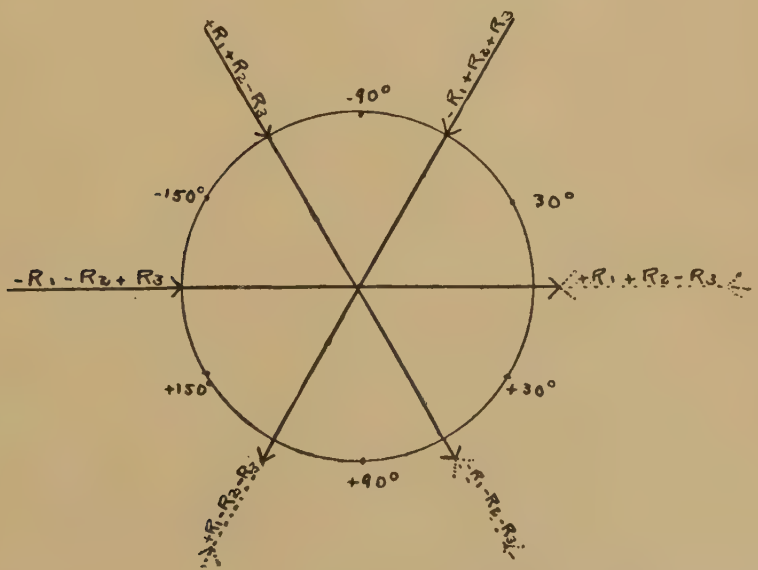


FIG. 299.—Diagram showing the direction of the leads with current (electrical axes) at various angles. The circle is divided into six sectors at 60 deg. each. The direction of the currents is marked by the arrow heads, the ventricular deviations for the respective leads by R1, R2, R3.

By these computations and methods, accurate knowledge of the direction and size of the electrical axis and of the electrical balance of the heart is obtained, but definite information regarding the origin of the excitation wave is not thus derived. A knowledge of the manifest size in conjunction with the electrical axis helps to diagnose the most likely point or points of origin of the excitation wave. By further amplification and applications of the Einthoven formulæ, Fahr and Weber have concluded as follows: The deviation Q results from excitation in the neighborhood of the middle zone of the heart; the papillary muscles and their environment. The apex of R is the resultant of excitation in the base of the heart, usually over its middle portion. The area is ordinarily to the left, but sometimes to the right of the middle line. The deviation S in general denotes apical negativity; the electrical center is usually to the left, but may be to the right of the median line. The deviation T is the end of the excitation wave. As a rule, it results from excitation of the ventricular base, to the right or left of the median line.

## Appendix[1]

### Table I

| α, degrees | Registered potential differences, e | | | Manifest potential differences |
|---|---|---|---|---|
| | $e^1$ | $e^2$ | $e^3$ | E |
| 0 | 10 | 5, 0 | − 5, 0 | 10, 0 |
| 10 | 10 | 6, 5 | − 3, 5 | 10, 2 |
| 20 | 10 | 8, 2 | − 1, 8 | 10, 7 |
| 30 | 10 | 10 | 0 | 11, 5 |
| 40 | 8, 2 | 10 | 1, 8 | 10, 7 |
| 50 | 6, 5 | 10 | 3, 5 | 10, 2 |
| 60 | 5, 0 | 10 | 5, 0 | 10, 0 |
| 70 | 3, 5 | 10 | 6, 5 | 10, 2 |
| 80 | 1, 8 | 10 | 8, 2 | 10, 7 |
| 90 | 0 | 10 | 10 | 11, 5 |
| 100 | − 1, 8 | 8, 2 | 10 | 10, 7 |
| 110 | − 3, 5 | 6, 5 | 10 | 10, 2 |
| 120 | − 5, 0 | 5, 0 | 10 | 10, 0 |
| 130 | − 6, 5 | 3, 5 | 10 | 10, 2 |
| 140 | − 8, 2 | 1, 8 | 10 | 10, 7 |
| 150 | −10 | 0 | 10 | 11, 5 |
| 160 | −10 | − 1, 8 | 8, 2 | 10, 7 |
| 170 | −10 | − 3, 5 | 6, 5 | 10, 2 |
| +180 | −10 | − 5, 0 | 5, 0 | 10, 0 |
| −170 | −10 | − 6, 5 | 3, 5 | 10, 2 |
| −160 | −10 | − 8, 2 | 1, 8 | 10, 7 |
| −150 | −10 | −10 | 0 | 11, 5 |
| −140 | − 8, 2 | −10 | − 1, 8 | 10, 7 |
| −130 | − 6, 5 | −10 | − 3, 5 | 10, 2 |
| −120 | − 5, 0 | −10 | − 5, 0 | 10, 0 |
| −110 | − 3, 5 | −10 | − 6, 5 | 10, 2 |
| −100 | − 1, 8 | −10 | − 8, 2 | 10, 7 |
| − 90 | 0 | −10 | −10 | 11, 5 |
| − 80 | 1, 8 | − 8, 2 | −10 | 10, 7 |
| − 70 | 3, 5 | − 6, 5 | −10 | 10, 2 |
| − 60 | 5, 0 | − 5, 0 | −10 | 10, 0 |
| − 50 | 6, 5 | − 3, 5 | −10 | 10, 2 |
| − 40 | 8, 2 | − 1, 8 | −10 | 10, 7 |
| − 30 | 10 | 0 | −10 | 11, 5 |
| − 20 | 10 | 1, 8 | − 8, 2 | 10, 7 |
| − 10 | 10 | 3, 5 | − 6, 5 | 10, 2 |
| 0 | 10 | 5, 0 | − 5, 0 | 10, 0 |

### Table II

Interpolation table

| Differences in degrees | | Potential differences +e | Differences in degrees | E |
|---|---|---|---|---|
| 0 | | 0 | 10 | 11, 5 |
| Leads I | 2 | 0, 4 | 8 | 11, 3 |
| | 4 | 0, 8 | 6 | 11, 1 |
| | 6 | 1, 2 | 4 | 11, 0 |
| | 8 | 1, 5 | 2 | 10, 8 |
| 10 | | 1, 8 | 0 | 10, 7 |
| II | 2 | 2, 2 | 8 | 10, 5 |
| | 4 | 2, 5 | 6 | 10, 4 |
| | 6 | 2, 9 | 4 | 10, 3 |
| | 8 | 3, 2 | 2 | 10, 2 |
| 10 | | 3, 5 | 0 | 10, 2 |
| III | 2 | 3, 8 | 8 | 10, 1 |
| | 4 | 4, 1 | 6 | 10, 1 |
| | 6 | 4, 4 | 4 | 10, 0 |
| | 8 | 4, 7 | 2 | 10, 0 |
| .0 | | 5, 0 | 0 | 10, 0 |

[1] These tables and the examples in the text were taken from Einthoven, Fahr and de Waart "Ueber die Richtungen und die Manifest Grösse der Potentialschwankungen, . . . des Elektrokardiogramms." (Arch. f. d. ges. Physiologie, 1913, CL, p. 308.)

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## CASE 1.—TYPICAL MITRAL REGURGITATION WITH DRY PERICARDITIS

H. R., female, aged eight, has never had scarlet fever, rheumatism or growing pains. There is no definite history of tonsillitis or of colds. Seven months ago she was supposed to have had "stomach trouble." She has had occasional nose-bleeds. Of late she has been short of breath.

**Examination.**—You see, gentlemen, that the child is well developed but is somewhat anemic. The systolic blood pressure is 90; the diastolic, 60. Inspection of the neck reveals nothing abnormal. Upon observing the chest you immediately notice the diffuse, precordial heave caused by ventricular systole. In addition to diffuse ventricular activity, palpation also reveals a distinct systolic thrill. There is no pain on pressure over the precordium. On auscultation you hear a loud, rough systolic murmur over the mitral area; this is transmitted to the axilla and over the entire back posteriorly. There is also a superficial musical murmur heard over the lower precordium and axilla. There is a soft systolic murmur over the aorta. The liver is not palpable. There are a few decayed teeth. The tonsils are enlarged. The urine is normal.

In this orthodiascopic tracing, you see an enlarged aorta; the heart is enlarged in a downward direction. The fluoroscopic examination does not reveal any fluid in the pericardial sac.

I present this case because the physical signs are so characteristic of a mitral regurgitant lesion with dry pericarditis. This combination is particularly frequent in children. In them pericarditis is often an accompaniment of a rheumatic exacerbation; it is apt to disappear with improvement or recession of the latter.

In spite of the absence of any definite history of tonsillitis or rheumatism, the great probability is that the child is suffering from the usual type of rheumatic endocarditis. Still, the original infection may have been a slight forgotten tonsillitis or even a catarrhal pharyngitis—the latter can occasionally (although rarely) be the starting point of endocarditis.

**Therapy.**—For the present, the child should be kept from school but she need not be confined to her room. She should have as much fresh air and sunlight as possible. She should be given substantial, fattening diet, including a fair proportion of meat. This is also a good time to have her tonsils and decayed teeth removed because the lesion is in a quiescent state. The final prognosis depends upon the frequency and the severity of future rheumatic or tonsillar attacks. If these be entirely absent or very infrequent within the next two or three years, the chances are that the endocardial lesion will remain quiescent. Under such favorable circumstances, one may promise this child a fairly active and useful life, as far as her cardiac condition is concerned.

### CASE 2.—TYPICAL MITRAL REGURGITATION AND PERICARDITIS; RE-EXAMINATION SIX MONTHS LATER; REMARKS ON TONSILLECTOMY AND EXTRACTION OF TEETH

H. F., female, aged eight, has had frequent attacks of tonsillitis, "growing pains," and articular rheumatism. She has also had nosebleeds occasionally. The family physician informed me that she has had a loud endocardial murmur for at least eight months. Three days before I saw her she had slight

fever, and painful and swollen knees. She now has shortness of breath and rapid, heart action. The last attack similar to this one occurred several months ago. The child's weight has been stationary for one year. She has been kept on a strict meat-free diet.

**Examination.**—The child you see is thin and anemic. Upon inspection, rapid heaving and regular ventricular action is plainly discernible. There is no pain upon palpation over the cardiac area. The apical impulse is reduplicated, and is felt most prominently in the fifth interspace, three inches from the mid-sternal line. On auscultation, the second pulmonic sound is accentuated. Over the mitral area you hear a loud, rough systolic murmur, which becomes more superficial and more musical toward the apex. The systolic murmur is also heard posteriorly. The aortic sounds are normal. The border of the liver is felt one and one-half inches below the free border of the ribs. There is no edema of the legs. The urine is normal. The tonsils are enlarged and look fleshy. Some of the teeth are bad.

The endocardial lesion is doubtless mitral regurgitation. The localized superficial musical murmur in the apical region can probably be regarded as indicative of dry pericarditis. Remember that typical to-and-fro friction sounds and pleuro-pericardial friction rubs are often absent in pericarditis. The only indication may be the superficial murmur, such as you hear so markedly in this case. The child is evidently suffering from an exacerbation of endocarditis due to the last attack of rheumatism. The pericarditis seems also due to rheumatism, for this lesion is often a superadded though temporary one.

The treatment I should suggest is rest in bed for about two weeks, and icebags to the heart when the action is rapid and violent. Salicylates are also indicated. The question of tonsillectomy and extraction of teeth I should hold in abeyance until the lesion has reached a quiescent stage. I consider it of prime importance to fatten the child. Starchy and fatty foods should be given, as well as some meat. If we succeed in fattening and strengthening the child, the chances are that she will be more resistant to future infections. I do not believe that meat should be excluded from the dietary for I have never been able to discover any connection between a diet containing meat proteins and rheumatic manifestations. As a matter of fact, this child has not had meat for months, yet she is now suffering from a fresh rheumatic endo-pericardial process. I believe that over-eating of meat can produce faulty metabolism, and can cause various so-called rheumatic manifestations, but I see no comparison between such cases (so-called rheumatism of metabolic origin) and actual bacterial infection, for that is probably the nature of rheumatic endocarditis.

**Examination Six Months Later.**—The history since the last examination states that there has been no epitaxis or rheumatism. The child's appetite has improved. She is now able to play with other children, and to go to school. The mother says that there has been no shortness of breath or rapid

heart action. The child's color is now good. There has been a decided gain in weight.

In this present examination you observe that the heart action is of normal rapidity when the child is at rest; there is a slight but normal temporary increase of cardiac rate after walking. Upon palpation, the reduplicated apical impulse (so-called gallop rhythm) felt six months ago has disappeared. On auscultation, you hear the same loud mitral systolic murmur as formerly, but the evidence of pericarditis—the superficial musical murmur at the apex—has disappeared. The orthodiascopic tracing now being passed around reveals an enlarged, somewhat ball-shaped heart. This contour is caused by an exaggeration and rounding out of the pulmonary, left ventricular, and right auricular curves.

It is evident that the cardiac and general condition is much better than when we first examined the patient. Her general appearance is better; there is no dyspnœa or tachycardia. The chief difference in the auscultatory signs is the absence of pericarditis. This is important not only because of the possibility that a localized pericarditis may interfere with proper cardiac activity, but principally because it is definite evidence that pericardial infection has receded. Quiescence of the endocardial lesion may also be assumed, for, although the mitral regurgitant murmur is as loud as formerly, still the gain in weight, and attendance at school without symptoms show unmistakably that no new endocardial damage has occurred. This is of the utmost prognostic importance, for it means that despite the present permanent changes in the valves and musculature, there is good ground for hope that the lesion will not progress any further, and that rheumatic manifestations will become less virulent and frequent.

The child's tonsils are enlarged. They look fleshy. I believe that this is the appropriate time for their excision.

What should be our attitude towards tonsillectomy in patients with rheumatic endocarditis? I think the present popular attitude of removing every tonsil that looks diseased is far too radical. It does not sufficiently consider the fact that tonsils are rarely normal, for they are the lymphatic portals through which, because of their position, bacteria of the most diverse kind must filter. We should, therefore, use discrimination in our advice regarding tonsillectomy. Tonsils that are palpably diseased, as shown by their gross appearance, and are, or have been the site of frequent inflammation, require thorough removal at the earliest favorable moment. The most propitious time is not during an attack of acute or subacute endocarditis, but when that process has become quiescent.

I hold similar conservative views with regard to the extraction of teeth. Decayed teeth have been almost indiscriminately accused of being the medium for rheumatic and endocarditic infections. Bad teeth, of course, require proper dental attention, usually extraction. I do not believe, however, that extraction should be practiced more radically because a patient happens to

have heart disease. In my own experience, I have not yet discovered that people with diseased teeth are especially prone to heart disease. I have, for example, found endocarditis in children with sound teeth and gums, as well as in those with diseased ones.

### CASE 3.—COMPENSATED MITRAL REGURGITANT LESION WITH VARIABLE AUSCULTATORY SIGNS

J. R., female, aged 25, unmarried, was examined by me for the first time two years ago. The history was one of frequent attacks of tonsillitis, for which the tonsils had already been removed. She has had no cardiac complaints, and no cardiac disease was suspected until two days prior to my first examination. She then had a sudden attack of "palpitation" with dizziness and vomiting. She had been swimming and roller skating the previous day.

Examination at that time showed a systolic blood pressure of 140; the diastolic, 90. Inspection of the chest revealed nothing abnormal. On palpation, there was a thrill-like systolic impact at the apex; there was no pain on pressure. Upon auscultation, there was a blowing, systolic murmur over the lower precordium, increased with exercise. There was also a soft systolic murmur at the right base. Abdominal examination presented nothing abnormal. The urine and the knee reflexes were normal; the pulse was regular, the rate, 76 per minute. Roentgenological examination showed slight enlargement of the aortic and ventricular shadows.

I regarded the case as one of simple mitral regurgitation. Because of her nervous temperament, I advised her to leave the city and to rest for a few weeks. Upon her return, the mitral murmur was scarcely audible.

I have had occasion to examine her two or three times since then for tachycardial attacks following acute indigestion. The cardiac rate would reach 120, the heart action, regular. Within twenty-four hours after a good purge and an ant-acid powder, the attacks passed off.

With these previous physical findings and history in mind let us consider her latest complaint. Yesterday, she had her usual sick headache from indigestion. She felt cold and weak and had occasional palpitation. The heart beat fast and regularly for about half a dozen beats. This was succeeded by a phase of several slow and irregular beats due to sinus arrhythmia. The composite effect on the rhythm may be compared to a horse galloping for a few seconds, and then suddenly checked and slowed. Now let us listen to the heart. We hear a very loud, blowing systolic murmur not only over the mitral area but over the entire anterior surface of the chest; I wish to emphasize the fact that the patient has no fever, nor any signs of decompensation. In view of my past physical findings—a soft mitral, and faint aortic systolic murmur—it will be of interest to re-examine this patient in a few days. For the present, without further attempts at diagnosis or of the import of the

murmur we shall give her an ant-acid powder, and small doses of codeine for her headache.

**Examination Two Days Later.**—The patient says she feels better. She has no headache nor "palpitation." Upon examination the extremities are no longer cold. Instead of the loud systolic murmur over the entire heart, there is again the soft, faint systolic murmur over the right base, and a somewhat louder one over the mitral area. In fact, the murmurs are of the same intensity as those I found at previous examinations. The cardiac activity is again normal in rate and rhythm.

How shall we interpret these differences in the auscultatory findings? In the absence of fever, and because of the temporary nature of the attack, I believe we can exclude recrudescent or recurring endocarditis as their cause. We know that mitral systolic murmurs may be not only of organic, but also of non-organic (so-called "functional") origin. In the latter instance, we assume that they can be caused by abnormal degrees of dilatibility of the mitral ring. In our patient, such a dilatation seemed to have been produced by the rapid heart action; this presumably allowed more regurgitation through an already damaged valve, and thus increased the loudness and propagation of the mitral murmur. Regarding the loud aortic murmur present two days ago, unless we believe this to have been due to upward transmission of the loud mitral murmur, its cause is difficult to explain. Even assuming a dilated aortic ring, this could scarcely be productive of an aortic systolic murmur.

This case is another exemplification of the fact that although murmurs are sometimes of great interest, they may after all have but little bearing on prognosis and therapy.

### CASE 4.—MITRAL STENOSIS—TYPICAL PHYSICAL SIGNS—RHEUMATIC RECRUDESCENCE

This man, 32 years old, had rheumatism several years ago. He was then told that he had "heart trouble." Except for occasional palpitation, he had no symptoms and was always able to work uninterruptedly at his trade as clothing cutter. About three months ago he caught a "cold," as he expresses it, which later developed into severe pains in the right thigh and leg. At about the same time he began to complain of shortness of breath, of palpitation and of slight precordial pains.

**Examination.**—You observe that the man is well-nourished, with a somewhat suffused cyanotic facies. Inspection shows exaggerated ventricular action. The pulse and heart action are regular: The rate, 110 per minute. Upon auscultation and palpation, you find at the apex the typical signs of a mitral stenotic lesion: A diastolic thrill both audible and palpable. The reduplicated second sound, rather typical of mitral stenosis, is absent. The liver is not enlarged, there is no edema of the legs. The blood pressure is normal. The urine is normal.

To judge from the hospital records and from the description of the patient's pains in the thigh he suffered from rheumatic sciatica. What caused the sudden onset of cardiac symptoms after years of quiescence? It was doubtless the rheumatic recrudescence, of which the sciatica was evidence, that caused the old valvular lesion to flare up.

Such recrudescences, actually re-infections, when severe, are often marked by irregular fever, by dry or serous pericarditis, by tachycardia, by dyspnœa, and by precordial distress. With continued infection and with changes in the cardiac musculature as well as in the endocardium, the patient begins to show some of the signs of decompensation: Enlargement of the liver, constant dyspnœa, brouchitis, edema of the legs, etc. In this stage, auricular fibrillation (q. v.) is apt to occur in mitral stenotic lesions, and with it, the special physical signs characteristic of that condition.

The remarks just made refer to severe re-infections. In milder rheumatic recrudescences, fever and other evidence of severe infection may be absent, or may be slight and fleeting, and, as in this patient, the cardiac complaints may be comparatively mild. Such cases are usually improved by salicylates. The patients should be kept quiet, although it is not necessary to confine them to bed for an inordinate length of time. Some physicians still believe that rest in bed for several months is necessary. In a mild case such as this, two or three weeks in bed are sufficient. The patient should then be allowed to walk around, and, if his occupation be not a very laborious one, he should then gradually retrain himself for his work.

**Question.**—How long does it take for mitral stenosis to develop?

Dr. N.—That depends a good deal upon the severity, the frequency and upon the site of the infection. It is the general assumption that it requires more than one attack of endocarditis to produce mitral stenosis. One may state in a general way that it takes several years for the development of marked mitral stenosis.

**Question.**—Is it possible to "clear up" an endocardial lesion with salicylates?

Dr. N.—The effect of the salicylates on the endocardium is still in dispute. Their action, in part at least, is to counteract the toxic effect of the rheumatic poison on the endocardium and on the joints. Since we possess no other specific at present, I deem it advisable to use salicylates in all cases of rheumatic endocarditis during the acute or subacute stages. I am in the habit of prescribing salicylate of soda in one gramme doses with compound syrup of sarsaparilla as the vehicle, given hourly, until six doses are given or tinnitus occurs. I then decrease the dose.

### CASE 5.—ACUTE RHEUMATIC ENDOCARDITIS—HEMIPLEGIA—DEATH

M. L., aged 16, had always been well until several months ago. She then developed a typical attack of acute articular rheumatism, with involvement of the larger joints and high temperature. An endocardial murmur was

heard for the first time several weeks ago. With one remission the temperature remained around 103° for eight weeks. There were irregular chills, anorexia and loss of weight. The pulse rate varied between 120 and 140 per minute.

**Examination.**—The patient looks emaciated. She says she has lost 25 pounds since her illness. The systolic blood pressure is 130; the diastolic, 80. Her face is flushed. You note that there is very little dyspnœa. You observe the rapid and regular ventricular action through the chest wall, and the rapidly pulsating aorta in the jugulum. Upon palpation, the ventricular impulse seems diffuse, although the cardiac outline is normal on percussion. Such impression of decided enlargement is frequently caused by a rapidly beating or overacting heart, although there may be no actual enlargement. I have often been able to demonstrate that fact fluoroscopically. Upon auscultation you hear a loud, rough systolic murmur over the lower left half of the chest; it is also transmitted posteriorly on the left side. There is a soft systolic murmur over the aorta. There are no signs of pericarditis. The lungs are normal. The heart is regular, its rate is 120 per minute. There are no petechiæ. There are at present no rheumatic articular manifestations. The liver is not palpable. A blood culture and blood count have not yet been made. The urine and blood pressure are normal.

There is, of course, no doubt as to the anatomical diagnosis. The patient is suffering from an acute endocarditis affecting the mitral valve. The important question to decide is whether the endocarditis is of the ordinary, so-called rheumatic type, or of bacterial origin. By bacteria, I mean streptococci, with actual recovery of the organism from the blood. It must be remembered that one, or even several negative blood cultures do not exclude the possibility of a streptococcemia. Since the blood culture was negative in our case, we shall have to arrive at a tentative diagnosis by studying the general clinical manifestations, and by searching for the usual characteristics of a streptococcus viridans bacteremia. We must search for petechiæ, for renal involvement (especially for the presence of microscopic blood in the urine), for a marked leucocytosis and for the usual signs of a severe sepsis: Delirium, chills, gastric symptoms, etc. The signs and symptoms just enumerated are present only in the more severe and acute forms of a streptococcic viridans infection. Even then remissions lasting weeks, or, rarely, months, may occur. Indeed, in cases in which the streptococcus viridans has actually been recovered from the blood, remissions of many months or years have been encountered. It is, of course, a question of terminology as to whether such long quiescent periods should be called "remissions." I have, for example, observed one case in which, after a violent course of several weeks, and with streptococci in the blood, the patient became well. The patient re-entered the hospital one year later, with a different complaint: He had been well in the interim. It has been stated that certain pathological characteristics of the heart and kidneys unquestionably demonstrate that

such patients have had healed lesions from an actual streptococcic viridans infection. A little consideration of this statement will show that this is by no means improbable. The long-continued infection, and the re-invasions so frequent in streptococcus viridans, are due to the washing into the blood stream of infected material and bacteria from the heart valves. Should these valvular vegetations be so protected or so favorably situated that the bacteria cannot be readily washed into the blood stream, it seems reasonable to assume that an occasional case of an actual streptococcic invasion will not only be quiescent for a long interval, but perhaps give time for actual healing of the infected valves.

To return to our patient, although the onset of the disease is violent and this patient is suffering severely from the endocarditic infection, the absence of petechiæ, of chills, and of delirium make me inclined to consider this case one of rheumatic endocarditis of a particularly virulent type. This is, of course, of great prognostic importance, since the chances for recovery from such an infection are greater than from a streptococcemia. Perhaps, also, the anti-rheumatic remedies—the salicylates—will be of help in controlling the symptoms. I believe the patient should get enough bromides, and, if necessary, opiates, to relieve her restlessness and quiet her heart action. Digitalis is not indicated because there is no decompensation.

**Later Report.**—A few days after you saw this patient, the family physician was suddenly called to her at her home. She developed hemiplegia, became unconscious and died a few hours later. It is evident that an embolus from the heart valve caused her death.

### CASE 6.—MITRAL STENOSIS—SUDDEN ONSET OF VASO-MOTOR SYMPTOMS

F. F., aged 38, married four years, has never been pregnant. She gives no history of rheumatism or tonsillitis. For some years she has had "heart burn" relieved by eructations. She has also complained of occasional palpitation and of shortness of breath when climbing stairs. She is able to walk well upon level ground. Three weeks ago, while walking against a strong wind, she developed severe mid-sternal pain. Since then, she feels as if she were unable to breathe freely and deeply. This, and the mid-sternal pain awaken her at night.

**Examination.**—The systolic blood pressure is 120; the diastolic is 70. Inspection of the chest reveals nothing abnormal. On palpation you feel a soft presystolic thrill in the apical region. On auscultation, you hear the typical presystolic thrill of mitral stenosis, and a somewhat accentuated second pulmonic sound. The knee reflexes are lively. There are no signs of decompensation. The urine is normal. The orthodiascopic examination shows a heart practically normal in outline.

The history is definite that the onset of the cardiac symptoms directly followed walking against a strong wind. Prior to that, there were very few,

if any, cardiac symptoms. One may of course dismiss this case as one of hysteria. But that diagnosis, so readily made and so often a cloak for inexact knowledge, will scarcely apply here. The patient states that the attacks occasionally awaken her from a sound sleep; that does not sound like hysteria. You also doubtless recall similar patients who otherwise bore their cardiac disease for years without pain or discomfort, in whom some inadvertance as fright, excitement or unwonted physical effort initiated cardiac symptoms. I believe we are here dealing with a similar instance. The occasional breathlessness is real, not imaginary. Its explanation however, is difficult. I believe it is of vaso-motor origin, and probably depends upon transitory changes in the vaso-motor center. One of my reasons for this belief is the beneficial effect produced by the extract of suprarenal gland in such individuals; it seems to have an immediate, although only temporary effect in restoring vaso-motor tone. Another possible cause of the breathlessness may be slight evanescent changes in ventricular dilatibility, which may also conceivably be accompanied by dyspnœa. With our present limited knowledge, this supposition is not susceptible to proof.

Therapeutically, I would advise suprarenal gland extract, in 2 grain doses, to be given during the attacks. I should also administer mixed bromides in 15 grain doses three times daily for several days. Depending on its effect, it may later be given in smaller doses. The patient should be reassured and told that she will positively improve, at first slowly, and then more rapidly. It will probably be several months before the symptoms disappear entirely.

## CASE 7.—MITRAL STENOSIS—PULMONARY INFARCT—GASTRIC AND VASO-MOTOR SYMPTOMS

M. A., aged 30, married, has had two children. Both confinements were normal. There is no antecedent history of tonsillitis, scarlet fever or rheumatism. Her baby is now nine months old. She had no symptoms which pointed directly or indirectly to her heart until three weeks ago. She then had an attack of hemoptysis. Since then she has frequent sudden attacks of an "empty" and "gone" feeling in the abdomen, and of precordial pains. Her pulse, the family physician tells us, has always been regular; its rate, 70 per minute. There has been no bronchitis. The patient has not vomited; she has no desire for solid food.

**Examination.**—You note that the patient is well nourished; that her face is flushed, and that she has no dyspnœa. Examination of the lungs shows the absence of rales and of other signs of pulmonary tuberculosis. An examination of the sputum one week ago showed the absence of tubercle bacilli. Inspection of the precordium and neck reveals nothing abnormal. Palpation and auscultation present typical signs of a mitral stenotic lesion: There is a palpable presystolic rumble and a loud presystolic murmur, both

most prominent in the apical region. Percussion shows that the cardiac outlines are within normal limits. The liver is not palpable; there is no edema of the legs; the urine and blood pressure are normal.

I present this case to you for several reasons. First, because it exemplifies a typical valvular lesion without a definite history of rheumatism, sore throat, or scarlet. However, the patient may have had "growing pains" in childhood, pains so mild in nature that she has long since forgotten them; a mild scarlet may have entirely escaped attention, or one slight attack of tonsillitis may have been the original source of the endocardial infection. We must also remember that patients of different types give varying heed to pains and symptoms, and what one may regard as a serious ailment, the other disregards entirely.

The second reason for presenting the case is that the patient did not know she had heart disease until the attack of hemoptysis, which was undoubtedly caused by a pulmonary embolism from the mitral stenotic lesion. The patient is not suffering from decompensation but since the hemoptysis, various precordial, gastric and vaso-motor symptoms have been present. Whether the cause of the infarct is to be sought in renewed endocardial infection or in the unfortuitous breaking off of an endocardial vegetation, I am not prepared to state. I am inclined to the latter view, however, because there are no symptoms—fever, sore throat, or rheumatism—suggestive of re-invasion. I do not know the exact nervous mechanism involved in the production of precordial pains in this case, but it has been my experience that any "insult" or renewed damage to a quiescent cardiac lesion can give rise to precordial discomfort accompanied by vaso-motor, gastric and other referred symptoms. I shall later point out that there exists an intimate relationship between the stomach and heart nerves, and that excitation of the one set often produces excitation in the other. I do not think that the patient is suffering from a primary gastritis; the sudden hunger spells, and the feeling of emptiness are probably reflex phenomena due to excitation of nerves of the stomach. One must also think of the possibility of an embolic infarct in the gastric vessels as a cause of the stomach symptoms. I believe, however, that it can be here excluded because there is no local epigastric sensitiveness nor has there been any hematemesis. The vaso-motor phenomena—flushes—are probably also of reflex origin. I recognize that these statements are not susceptible to proof; too many links in the chain of evidence are missing; hence I do not wish my theories taken too dogmatically. On the other hand my experience with other similar cases makes me feel that the explanation given, although hypothetical, is essentially the correct one.

The treatment and prognosis in this case are of interest. When vaso-motor symptoms of reflex nature (especially when associated with precordial pains) are prominent, I have found the solid extract of the suprarenal gland in one or two grain doses, administered three times daily, to be of great value. The gastric symptoms can usually be controlled by giving half a teaspoonful

of an alkaline powder containing equal parts of magnesium usta and bicarbonate of soda after nourishment. Food should be light, should be given frequently and in small quantities. Acids, and heavy and greasy meals should be avoided. It may be necessary to follow a strict "ulcer diet" for a week or two. Digitalis is of no value in this case, and should not be advised. The patient should be kept in bed for about a week or two, until the precordial and gastric symptoms subside. Later, she may gradually return to her previous mode of life and diet. I emphasize "gradually," because if too much is undertaken at first, the symptoms are apt to be aggravated, and convalescence correspondingly retarded.

### CASE 8.—MITRAL STENOSIS—ATYPICAL HISTORY AND SYMPTOMS

S. A. male, aged 58, states that about four years ago he had "rheumatic" pains in the shoulder; these lasted several weeks. He does not remember any previous attack. He does not know whether he ever had scarlet fever. He had no cardiac complaints until several months ago. The initial symptoms were pains in the left chest and some dyspnœa upon walking. He also developed gastric symptoms; these consisted of sticking pains in the left, and occasionally in the right breast, one-half hour after his meals; his hands would then become cold. None of the usual symptoms or signs of indigestion were present; no nausea, anorexia, epigastric distress, distension or coated tongue.

A more exact history of the onset of the very first rheumatic attack would have been of importance, for the patient's valvular lesion—mitral stenosis—could scarcely have developed, according to his story, within four years, without repeated and severe rheumatic attacks and endocardial recrudescences. The assumption, therefore, seems fair that there had been other rheumatic manifestations, overlooked or forgotten by the patient, and antedating the distinct rheumatic attack of four years ago.

**Physical Examination.**—You note that the patient is somewhat dyspnœic when lying down. Upon inspection of the chest you observe the slight ventricular hyperaction confined to the apical region. Upon palpation you feel the typical diastolic thrill so characteristic of advanced mitral stenosis. On auscultation you hear the rumble occupying almost the entire diastole and apparently increasing in intensity as it approaches the systole. Following the systole, you hear a reduplicated second sound characterized by a sharp, valvular double click. In brief, all the classical signs of a mitral stenotic lesion are present. I would remind you, however, that you must not expect all the text-book signs of valvular disease in every case; if you do you will be doomed to frequent disappointments.

Of great clinical interest is the advanced age of this patient, for it is very rare to observe a patient over 50 with mitral stenosis, especially with a normal,

rhythmical pulse. Of even greater rarity is the first appearance of any cardiac symptom of this lesion so late in life.

Let us now attempt to correlate the symptoms following the ingestion of meals with the chest pains. You recall that there exists a ganglionic connection between the gastric filaments of the vagus and the nerves of the heart. The exact nerves forming the reflex arc are not known, but we possess many clinical observations which demonstrate that cardiac symptoms, *e.g.*, dyspnœa and precordial pains, otherwise slight, are often aggravated during meals or during digestion. Such chest pains are currently attributed to pressure exerted by an overdistended stomach against the diaphragm and thus against the heart, with consequent circulatory embarrassment and precordial pains. I have made a fluoroscopic study of the stomach in some patients with cardiac disease who suffer from the symptoms I am describing. In no instance in which I gave opaque meals of bismuth or barium, did I find evidence of any undue amount of gastric distension or of pressure against the diaphragm. I therefore conclude that neither gastric overdistension nor pressure is a factor in the production of precordial pains after meals in these individuals. I believe that the precordial pains are caused by excitation of cardiac nerves or plexuses that are reflexly stimulated by the gastric filaments during the process of digestion. We know that the heart does not contain nerves of sensation, but, according to the theory of Head, we assume that corresponding spinal segments can be reflexly excited by peripheral irritation from an end organ, in this instance, the heart. The spinal segment or segments involved then produce centripetal impulses along the spinal nerves supplying the skin and muscles of the precordium, which the patient feels as pain. To use an analogy, I have observed that, similar to the hypersusceptibility found in anaphylaxis, the cardiac nerves and spinal segments are frequently sensitized, so to speak, a condition which makes them hypersusceptible to any neurogenic influence, normal or abnormal. To apply these considerations to our patient, the vaso-motor symptoms (cold hands) and the chest pains, both occurring during the process of digestion, are probably due to reflex excitation, in the former, of the vaso-motor center, and in the latter, of a spinal segment.

**Therapy.**—There is no actual decompensation nor any evidence of endocarditic recrudescence. The cold hands, precordial pains, and dyspnœa after meals seem due, according to my assumption, to reflex disturbances in a "nerve-sensitized" individual with a mitral stenotic lesion. I do not believe digitalis is indicated. I shall advise the patient to take a cup of warm milk or hot coffee in bed before arising. He should rest for about one hour after meals. If the symptoms do not improve, I shall advise him to take one or two teaspoonfuls of aromatic spirits of ammonia in water at such times when symptoms occur. This simple remedy seems to act favorably by its effect upon the nerve filaments of the stomach.

## CASE 9.—MITRAL STENOSIS—HYPERTENSION—NEURASTHENIA

Mrs. F., aged 32, had chorea as a child. She has had "palpitation" for many years, especially when walking up stairs or when nervous. The palpitation is occasionally accompanied by a tight feeling across the chest. The patient has had frequent crying and hysterical spells. She has two children. There are no cardiac symptoms during sexual intercourse. She has had a good deal of trouble with servants of late, a matter which has upset her and brought on attacks of crying. She has always been stout; there has been no loss of weight in recent years.

**Examination.**—This patient, as you notice, is plump and florid. She is even now having a crying spell. This has the effect of causing a pulse rapidity of 110 per minute. Immediately before crying, the rate was normal. It seems probable, therefore, that the "palpitation" of which the patient complains is due to tachycardia. There is no dyspnœa when she is sitting or lying down. Examination of the mouth shows that many of the teeth have been filled. Inspection of the chest reveals nothing abnormal. On palpation and auscultation, a typical presystolic thrill is felt and heard. There is no pain upon precordial or epigastric pressure. You hear a reduplicated second sound over the cardiac apex and over the pulmonic; it is more intense at the latter site. Over the aorta, there is a very soft systolic murmur which accompanies the first sound; the second sound is somewhat accentuated. Examination of the abdomen reveals nothing abnormal. The urine is normal. The knee reflexes are normal. There is no edema of the legs. The blood pressure reading is surprisingly high; the systolic is 230; the diastolic, 110. The orthodiascopic tracing being passed around shows a somewhat enlarged aorta. The heart lies rather flattened upon the diaphragm; it is slightly enlarged to the left, as shown by the X-ray.

There is no doubt as to the cardiac lesion. The patient has a typical mitral stenosis. In the absence of any unwonted exertion or excitement, however, she has no cardiac symptoms. Indeed, I understand from her family physician, that it is only very recently that she was thought to have heart disease. The lesion is undoubtedly a quiescent one, for the cardiac symptoms are due entirely to the strain of domestic trouble with servants in a pampered individual. It shows again how extraneous circumstances can affect a heart lesion and can give rise to symptoms. This patient, for example, might have continued for a very long time with no more than her previous symptoms of occasional tachycardia from stair climbing. She belongs to the type of women who are easily ruffled and upset when their menage does not run smoothly. They are not the individuals who can "pitch in" and do things themselves. They become restive and nervous, a condition which deleteriously reacts upon various mechanisms under the control of the nervous system. You recall the extremely high blood pressure in this patient, yet there is nothing in the cardio-vascular examination to suggest a pathological organic cause for this

extreme hypertension. The arteries are not thickened; the urine is normal; there is no edema. A pheno-sulphophthalein test has not as yet been done, and it would be hazardous to diagnose this hypertension as being entirely of neurotic origin upon only one blood pressure reading, but I shall be much surprised if further observation does not show lower and even normal blood pressures. Indeed the crying itself may have caused the hypertension.

*Therapy* consists chiefly in reassuring the patient and in getting her away from her home environment for a while. A short vacation of a week or two would be of great benefit. Bromides given in moderate doses for several days would also be of value in tiding the patient over the critical stage of her nervousness.

### CASE 10.—RHEUMATIC ENDOCARDITIS IN A SYPHILITIC

Mrs. W., aged 38, has two children. She has had several miscarriages. At various times she had luetic ulcers in the mouth and luetic skin lesions. She has been on mixed treatment for a long time. Although frequently "nervous" and suffering from fatigue, she never had any actual rheumatic manifestations until four years ago. She then had an attack of tonsillitis followed by lumbago. Several months thereafter she had a stiff neck, "blood spots" over her body (probably purpura hemorrhagica) and two severe attacks of migraine. One year later she had a "cold" followed after four weeks by irregular chills and by pains in the lower right hypogastrium. Shortly thereafter she suddenly developed sharp epigastric pains with chills, vomiting, fever and jaundice.

When I first examined her, two days after her attack, the jaundice had disappeared. She was dyspnœic; the temperature was 101°. Pressure over the epigastrium caused pain which radiated to both lumbar regions. There was no pain over the appendix or gall bladder region.

**Cardiac Examination.**—Inspection and palpation of the precordium revealed nothing abnormal. The cardiac area also seemed normal to percussion. Auscultation at the apex revealed a loud systolic murmur which was transmitted laterally to the left axilla. There was a localized pericardial friction rub heard over the apical region. The throat was normal. There were no purpuric spots on the body. There was no edema of the legs. The urine and blood pressure were normal. The Wassermann blood examination was positive. Several months later the patient developed nose bleeds and an attack of erythema nodosum.

**Present Examination.**—Despite the stormy and typical rheumatic history lasting over one year, you note that the patient is at present in excellent condition. She is not dyspnœic; she says she can again take up her household work; she can walk well without losing her breath. There is still a loud systolic murmur over the mitral area. There are no signs of pericarditis or decompensation.

There are two points of especial interest in the previous history: Syphilis and rheumatic infection. In view of the definite and typical rheumatic history and of the present typical mitral regurgitant lesion, and because of the absence of any active syphilitic lesion during the course of the rheumatic attack, I have no doubt that the endocarditis is of rheumatic origin. Further evidence of this is the absence of aortic involvement, the usual cardiac affection in syphilis. The endocarditis has reached a quiescent stage; this probably accounts for the excellent cardiac condition of the patient. Mitral regurgitant lesions, when quiescent and not re-infected, are especially prone to allow the heart to carry on the circulation almost normally, despite crippling of the valve. Such a favorable status naturally also depends upon the condition of the musculature. For example, if there be extreme ventricular hypertrophy, or if upon clinical grounds, there is assumed to be infiltration of the myocardium with submiliary nodes (Aschoff's bodies), cardiac power will suffer, cardiac reserve power will be diminished, and dyspnœa and other signs of heart failure will appear. Questions regarding prognosis in cardiac diseases always involve not only the amount of damage and the cardiac status, but also the state of activity of the lesion. The latter cannot usually be decided by physical signs alone. In the case before us, for example, the physical signs indicate simply a rheumatic affection of the mitral valves. The history, however, becomes a fair prognostic guide. You recall that endocarditis began after many months of rheumatic manifestations. There have been no rheumatic or cardiac symptoms since that time. There is at present no decompensation; the heart is apparently not enlarged. In other words, we must assume that the endocardial infection has been inactive for some time, that the damage to the musculature has been minimal, and that the cardiac power is practically normal. There must of course be some reservation as to what might happen to this heart in the future. From my experience with similar cases, I believe the main danger lies in the possibility of a rheumatic endocarditic recrudescence. Such a process often lights up dormant and quiescent lesions. It may then rapidly lead to death either by causing embolic infarcts into important organs, or more slowly, by initiating a progressive train of symptoms, from mild myocardial insufficiency to fatal heart failure. On the other hand should no infection occur, the patient may live comfortably and perhaps as long with, as without, the mitral lesion. I do not believe that a luetic infection superimposed upon the present lesion need be feared, in spite of the positive Wassermann reaction. The latter simply means that there exists an active luetic focus somewhere in the body. Were the heart the site of this focus, evidence of an aortitis ought to be present by this time.

Regarding therapeusis, I have at no time given the patient digitalis, because decompensation was never present. Besides, digitalis is rarely of value when the heart is the seat of an active inflammatory process. Main therapeutic reliance was placed upon the salicylate of soda which I gave at

first in 15 grain doses hourly for 8 doses, and then in decreased quantity and at longer intervals. When the heart action was rapid I gave codeine, combined with the bromides alone or together with chloral. In other words I attempted to treat the infection with the drugs at our command. I say "drugs" advisedly, because I have seen no beneficial effects in similar cases from stock or autogenous vaccines of various kinds, nor from the use of silver salts intravenously, as more recently recommended. Rheumatism is very probably of bacterial origin, although the specific organism has not yet been positively identified. And if the bacterium be a streptococcus, as is generally assumed, we have no grounds for believing in the present state of our experience with vaccines that they will in any way influence, control, or curtail the infection.

### CASE 11.—DOUBLE MITRAL LESION—ACUTE PERICARDITIS WITH EFFUSION—CLASSICAL SIGNS AND SYMPTOMS

R. W., male, aged 16, has had "heart trouble" for ten years. The only cardiac symptom during the entire time was occasional dyspnœa when running. He has had attacks of articular rheumatism every spring. One week ago he had a sore throat accompanied by pain in the legs. A day thereafter he had severe dyspnœa, chills, fever and pain in the right nipple region when breathing.

**Examination.**—You note that the boy is orthopnoic; he has to sit straight in bed in order to breathe with any comfort. Upon inspection, you observe the diffuse precordial heave and the throbbing carotids. Upon palpation, you feel a typical presystolic thrill at the apex, indicative of mitral stenosis. Upon percussion, I want you especially to note, besides enlargement of the entire cardiac area to the right and left, the small but definite area of dullness in the second and third left interspaces. Upon auscultation, there are heard a loud systolic, and a rough and rasping presystolic murmur. In addition, you hear the to-and-fro friction rub over the lower sternum, the classical sign of pericarditis. The liver is not palpable. There is no edema of the legs. The pulse is regular, the rate, 120 per minute. The systolic blood pressure is 140; the diastolic, 80. The urine is normal. The throat and teeth are normal. The present temperature is 103°.

I present this case in contrast to the last (Case 10), because of its many typical features. The history of regular attacks of rheumatism and tonsillitis, the typical signs of a double mitral lesion with pericarditis, the breathing, the sharp onset of the present invasion preceded by a sore throat, all are characteristic features of rheumatic endocarditis with an acute recrudescence. What is of especial interest, perhaps, is the area of dullness in the upper left interspaces already pointed out to you. That is most probably indicative of fluid in the pericardial sac. Fluid in the pericardium is by no means readily recognized; indeed, of all lesions, it most often evades our most painstaking efforts at diagnosis. It has been shown experimentally that at the beginning,

fluid accumulates at the base of the heart around the great vessels. When the effusion is massive, it may produce dullness in the upper left interspaces as in this case. More often, however, dullness cannot be elicited, even when the fluid begins to fill up the lower part of the sac. This may be because such fluid is apt to sag posteriorly instead of anteriorly; or because dullness caused by it cannot be distinguished from that of the heart itself. Perhaps differential dullness can be more readily elicited in those instances in which the heart is not enlarged, and the pericardial fluid is disproportionately massive. Even roentgenograms often fail to demonstrate the presence of fluid, for a definite double shadow—roentgenographic evidence of pericardial effusion—is by no means always present. Clinically, we must often rely upon the presence of the dry pericardial friction rub, and upon the course of the invasion, for our diagnosis. If the former is present, the invasion febrile and stormy, and the patient young, the great chances are that fluid in varying amounts is present in the pericardial sac. This is borne out by many autopsy examinations. In addition, we find clinically that dyspnœa is often present and that convalescence is prolonged, even when the physical signs of dry pericarditis have disappeared. In other words, when definite differential dullness cannot be elicited, we must be guided by the severity of the invasion, by the prolonged convalescence, and by the pericardial friction rub. The latter is rarely absent throughout the entire disease.

I believe that the boy will weather this attack of pericarditis despite its stormy onset. I shall put him on large doses of salicylate of soda for the next few days. I shall give him sufficient bromides, and if necessary, codein, in order to quiet his pain and dyspnœa. The probabilities are that the signs of dry pericarditis and of fever will disappear in several weeks. Blood cultures will also be made, although my past experience with many similar cases has been that the culture is negative. Of course, when this invasion is past, the signs of endocarditis—the double mitral lesion—will still be present.

### CASE 12.—DOUBLE MITRAL LESION—DECOMPENSATION—STREPTOCOCCEMIA

H. G., aged 15, was said to have had a "heart murmur" three years ago. There was no definite history of rheumatism, scarlet or tonsillitis. The boy had always been able to run and play until three months ago, when cardiac symptoms appeared for the first time. The main complaint was dyspnœa. The family physician told me that at that time the patient's liver was enlarged and that it pulsated. He vomited, but had no fever. His pulse was rapid, the rate 140 per minute. Two days ago petechiæ appeared upon the chest. He has been vomiting almost continually since then.

**Examination.**—You note that the boy is emaciated and orthopnoic. He is somewhat drowsy. You note also the enlarged and tortuous jugulars. You observe the tremendous diffuse precordial systolic heave of the entire lower and left lateral half of the chest, indicative of extreme ventricular

hypertrophy. The left side of the chest is much more prominent than the right. This in itself would indicate hypertrophy rather than dilatation. Upon palpation, you feel a diffuse pulsation and a distinct systolic thrill. Upon auscultation, you hear a loud double mitral murmur transmitted mainly to the left. You also hear a superficial friction sound in the second left interspace, indicative of dry pericarditis. The pulse rate is 70 per minute. The liver is enlarged, its lower right border is one inch below the umbilicus. You can also see the liver pulsate quite distinctly. There is slight edema of the legs. The urine is scanty and contains albumen and casts. The anterior surface of the chest is sparsely covered with little hemorrhagic spots which do not disappear upon pressure; these are petechiæ.

It is of course evident that the patient is suffering from extreme heart failure; the orthopnœa alone is sufficient evidence of that. If the facts of the history be correct—namely, that a heart murmur, and therefore presumably that heart disease existed for but three years, there has been exceptionally vast damage to the heart muscle in that comparatively short time, especially in view of the absence of cardiac symptoms until three months ago. The present streptococcic condition is demonstrated by the petechiæ, which can be regarded as undoubted clinical evidence of such infection. I emphasize clinical, for a single or even several bacteriological examinations of the blood may not recover the invading organism. Its recovery from the blood depends not only upon frequency of blood cultures but also upon the number of the invading organisms, and the latter can only be approximately guessed at by the severity of the clinical manifestations and the amount of petechiæ. Invasions with the streptococcus viridans is not unusual in severe endocarditis and is often a terminal event.

The drowsiness and vomiting may be nephritic in origin, the result of renal congestion or bacteremia. The renal condition may be aided by the administration of a Murphy drip of 5 per cent. glucose solution containing bicarbonate of soda; this would not only aid the kidney function, but would also supply fluid to the body and help combat a threatening acidosis. Digitalis may also be of some advantage. But because of the advanced heart disease with the added bacterial infection, I believe the case is hopeless and will probably end fatally in a short time.*

## CASE 13.—MITRAL REGURGITATION—CORONARY(?) INVOLVEMENT OF UNDETERMINED ORIGIN

**Gentlemen.**—We are fortunate in having the advantage of a careful history and the results of several prior examinations in this interesting case. The history is as follows:

R. R., male, aged 39, a dentist, is married and has two children. He had gonorrhea many years ago. A Wassermann blood examination taken seven

* The patient died within a week of this examination.

years ago was negative. He has never suffered from rheumatism and only very rarely from tonsillitis. Eight years ago he was operated upon for gangrenous appendicitis. Six years ago a gastro-enterestomy was done for duodenal ulcer. He has had no gastric complaints since the last operation. About two years ago he was accepted for an increase in life insurance, so that he very probably had no obvious signs of endocarditis at that time.

The cardiac history begins with the statement that about a year and a half ago a friend of the patient, a physician, noticed that his finger nails were somewhat cyanotic. The patient at that time, however, had no cardiac complaints nor did a cardio-vascular examination then made disclose abnormal or other signs. About one year ago and again eight months ago, the patient suffered from sudden severe midsternal pains radiating to the back. These attacks lasted several hours; they were not accompanied by dyspnœa. Again a cardio-vascular examination revealed nothing abnormal: No murmurs, no decompensation; the urine was normal; there was no fever. The attacks were regarded as being probably of gastric origin. In the interim between attacks and until several months ago, the patient felt perfectly well and was able to attend to his work. He then began to suffer from dyspneic attacks which, with intervals, have continued up to the present time. Coincident with the dyspnœa an endocardial murmur was heard for the first time. The first dyspneic attack was accompanied by a temperature of 101° which lasted several days.

The patient states that he cannot climb stairs or walk rapidly without having dyspnœa and tachycardia. Two or three months ago cultures from tooth sockets were made and the streptococcus viridans was isolated. Several teeth were extracted upon the theory that these had some relation to his cardiac disease. No improvement followed the extraction.

Now let us examine the patient. The man you observe is well nourished. When sitting quietly, dyspnœa is not noticeable. The systolic blood pressure is 120; the diastolic, 90. Upon inspection and palpation, you note ventricular overaction in the mitral area. The carotid and jugular pulsations are normal. There is no pain on precordial pressure. Upon auscultation you hear a loud, somewhat musical murmur over the lower precordium; it is transmitted to the left and is unaffected by respiration. The other sounds are normal. The orthodiascopic tracing, as you see, shows a normal-sized aorta. The left ventricular and right auricular curves are enlarged. The electrocardiogram of this patient is also of some interest and importance. Note that the rhythm is normal; all the deflections are small and the ventricular deflection in lead III is marked by being splintered and by its abnormal width. These characteristics are strongly suggestive of myocarditis.

Except for scars of previous operations the abdominal examination reveals nothing abnormal. The liver is not palpable. There is no edema of the legs. The knee reflexes are almost entirely absent. The pupillary reflexes are normal. There is no ataxia or Rhomberg symptom.

It is apparent that the patient is suffering from a mitral regurgitant lesion with heart failure: You observed, for example, that even when he walks slowly about the room dyspnœa becomes quite marked. The subjects of special interest are the etiology of the endocarditis and of the previous severe substernal attacks of pain, and the therapy to be applied.

I believe the usual causes of endocarditis—rheumatism and tonsillitis may be excluded in this individual. Another Wassermann examination should be made to exclude syphilis, although neither the history nor the type of lesion point to syphilis. Nor do I regard the teeth as a possible causative factor despite the discovery of the streptococcus viridans in cultures from tooth sockets. It has been demonstrated that this organism is by no means an infrequent harbinger in the mouths of persons with normal hearts. I believe the importance and frequency of tooth infection as a source of endocarditis have been tremendously overrated. And even in rare instances where this etiology can be definitely established, it is in my opinion still an open question as to how much good can be accomplished by routine extraction in an assumed hemotological infection which has already damaged the cardiac valves, although of course extraction seems indicated in such cases.

In view of the previous abdominal operations on our patients one must naturally attempt to correlate a possible post-operative infection with the present endocarditis. But the operations have so long antedated the cardiac symptoms, and the abdominal and gastric condition is now so satisfactory that, in the light of our present knowledge such correlation can only be regarded as hypothetical.

In occasional instances of marked cardio-sclerosis, the coronary arteries seem to be the first point of attack; and as evidence of this, the symptoms of such patients begin with attacks of precordial pain before other marked manifestations of cardiac disease present themselves. So also in this case, to judge from the history and subsequent course the first of the cardiac structures to be attacked were presumably the coronary arteries, for I believe the previous attacks of precordial pain must be regarded as very probably of coronary origin. Even the cyanosis of the finger nails, months before the actual cardiac symptoms began, may have been a precursor of disturbance of the circulation antedating more obvious signs and symptoms.

The prognosis must be regarded as exceedingly grave because we are probably dealing with a progressive, non-quiescent process; because of the more or less constant dyspnœa; and because of the significant evidence of probable hypertrophy and myocarditis revealed by the X-ray and electrocardiogram. Should the endocarditis subside or yield to therapy, the patient may be comfortable for a number of years.

Therapy, however, does not offer much to this individual. I would recommend a mixture containing the salicylate of soda and the mixed bromides with some simple syrup as a vehicle. The bromides are meant to quiet the heart action. The salicylates are given upon the possibility that the endo-

carditis is of rheumatic origin. I expect no benefit from its use here but I feel that it is due the patient to administer remedies even empirically, so long as they do no harm. Digitalis is not indicated just now because the dyspnœa is probably caused by insidious advance of the inflammatory process in the endocardium and myocardium. Yet its use can do no harm. Should a Wassermann blood test be strongly positive, we would then naturally have some assurance that appropriate antiluetic treatment would be followed by excellent results.

### CASE 14.—AORTIC REGURGITATION—TYPICAL PHYSICAL SIGNS

L. J., male, aged 26, married, for many years has had attacks of fever twice yearly, unaccompanied by catarrhal manifestations. He has had no sore throat nor cough. He is a moderate cigarette smoker. Aside from the fever spells and one attack of gonorrhea several years ago, he has always considered himself well. He has always been athletic and has never had any cardiac complaint, except occasional "pounding" of his heart when lying upon the left side. His work is now sedentary.

**Examination.**—The systolic blood pressure is 130; the diastolic, 25. You note that the patient lies down and walks without dyspnœa. You observe the overacting carotids and the over-pulsatile rise of the tissues in the jugulum. You observe also the strong ventricular impulse shown by overaction in the mitral area. There is no pain on pressure over the precordium. There is a suggestion of a palpable systolic thrill in the apical region. Upon auscultation, there is a loud diastolic murmur heard especially over the left base and transmitted downward. At the apex, there is heard a short, presystolic murmur. You likewise feel the typical collapsing, so-called Corrigan pulse; this becomes more evident by feeling the radials when the patient's arms are extended above his head. The abdomen presents nothing abnormal. The urine and knee reflexes are normal. There is no edema of the legs. The tonsils are small; the teeth are in excellent condition. A Wassermann blood reaction was negative; a complement fixation test for gonorrhea was also negative. The orthodiascopic tracing, a copy of which you see, shows that the aorta is slightly enlarged and that the left ventricle is enlarged to the left and below. It would have been interesting to have you observe the aortic over-pulsation in the fluoroscope, a condition which always gives the impression of a very much enlarged vessel.

I show this patient because the physical signs of aortic regurgitation are so characteristic. The murmurs of aortic regurgitation are occasionally so indistinct that, if the pulse be not of the characteristic collapsing type, a diagnosis of aortic regurgitation may be almost impossible. One may then compare the arm and leg blood pressures; in aortic regurgitation, the difference between the leg and arm systolic pressures is much more than in normal individuals.

The infection which produced the aortic lesion in this case is obscure. Clinically there is little reason to doubt that the semi-annual febrile outbursts have some etiological connection with the lesion, but their exact nature—whether of tonsillar, rheumatic or other origin—cannot be determined from the patient's description. Treatment for the present must, if possible, be preventative. I shall allow the patient some exercise, because the lesion is quiescent, there is no decompensation, nor are there any cardiac symptoms. I shall limit his smoking, because the latter may initiate abnormally rapid heart action or make the patient more likely to get precordial pains. I shall later explain my view of the effect of tobacco upon the heart. I shall allow the patient to play golf occasionally and to do a moderate amount of walking, but I shall warn him against running and excitement because of their possible effect in inducing rapid heart action. Once tachycardia, no matter from what cause, occurs in aortic regurgitant lesions, it becomes a troublesome symptom and is difficult and tedious to control.

Patients with aortic disease often do well until about their fortieth year. They then are apt to develop streptococcic viridans infections. I am as yet uncertain whether it may not be wise to vaccinate such patients with streptococcus viridans in their earlier years, in order to minimize this danger if possible. Stock vaccines would of course have to be used. A series of injections would probably have to be repeated every two to three years, the chances being that the immunity would not last longer than two years after each series of injections.

### CASE 15.—AORTIC REGURGITATION—MITRAL REGURGITATION—ACUTE DRY PERICARDITIS

**Gentlemen.**—This little girl is nine years of age. The mother says that the child had a distinct attack of articular rheumatism of two weeks duration when she was three and one half years old. There was no evidence of cardiac involvement then, the mother tells us, for the physician at that time carefully and regularly examined the child. Later the diagnosis of heart disease was made. As a consequence the child has been kept quiet and rarely allowed to romp about. Several months ago the child complained of headache and of pains in her hands and feet for several days. The little patient herself never made any complaint about her heart until about one week ago. She then had fever and said that her left side hurt her.

**Examination.**—You observe that the child is somewhat undersized and anemic. She is not dyspneic. You note the exaggerated ventricular action in the left nipple region. Because of the thin chest walls you may even observe the overacting pulmonary artery at the second left interspace. On palpation, you feel an exaggerated ventricular impact in the apical region and the click of pulmonary valve closure in the second left interspace. The sounds heard upon auscultation are confusing and require careful study for their proper interpretation, especially since the heart beats irregularly.

We shall disregard the arrhythmia for the present and attempt to unravel the various murmurs and adventitious sounds. Let us start with the mitral area. There we hear a systolic murmur which accompanies but does not replace the first sound; the murmur is transmitted slightly toward the left axilla. There is also a somewhat superficial friction rub heard at the apex at the extreme end of inspiration. In addition, there is a short presystolic murmur heard only over the left nipple. Over the right base the first sound is normal and the second is continued into a soft diastolic murmur. Over the left base the first sound is normal and the second pulmonic is markedly accentuated; this is the sound which caused the palpable click already referred to. The second pulmonic is immediately followed by a diastolic murmur somewhat louder than the one on the right side; the murmur tails away toward the cardiac apex. I believe we are dealing with two distinct valvular lesions; aortic and mitral regurgitation. The former has caused the diastolic murmur over the right and left base with transmission downward; the murmur is more distinct upon the left than upon the right side—a not unusual occurrence. The aortic regurgitation is also the cause of the short presystolic murmur at the apex, a so-called Flint murmur. You may ask, why may not the latter be caused by a mitral stenotic lesion? Several facts speak against this supposition. First, mitral stenosis is very rare in children. Second, there is absence of a palpable diastolic thrill at the apex. Third, there is no reduplicated second sound so common in mitral stenosis. There is something to be said as to whether the left sided diastolic murmur may not be due to relative insufficiency of the pulmonary valves. This produces the so-called Graham-Steele murmur which is also diastolic in time and is transmitted along the left sternal border. I believe however, the Graham-Steele murmur can be excluded because we have unmistakable evidence of closure of the pulmonary valves in the sharply accentuated second pulmonic sound.

Let us now study the arrhythmia. By listening at the apex and feeling the pulse, you note that every systole produces a corresponding pulse wave; that is, there are no pulse intermission or so-called "missed" beats. The heart pulsates at an increased rate for several seconds, then at an abnormally slow rate for three or four beats, to be followed by a pause comprising an interval of about two normal systoles. In this silent cardiac interval there are no jugular or carotid pulsations, a fact which you may determine by scrutinizing the neck. We may therefore exclude extrasystoles as well as the rarer arrhythmia known as blocked auricular beats. I believe that we are dealing with sinus arrhythmia, here sufficiently marked to occasionally block out an entire auriculo-ventricular sequence, *i.e.*, a complete heart beat. This is known as sinus block or sino-auricular block and is ordinarily but an extreme degree of sinus arrhythmia.*

* An electrocardiogram which I took some days later showed marked sinus arrhythmia.

**To Summarize.**—A child of nine with a typical rheumatic history only recently complained of left-sided precordial pain. Examination revealed pleuro-pericarditis, aortic and mitral regurgitation, sinus arrhythmia, and perhaps also sinus block. There were no signs of decompensation. Perhaps some of the physical signs will become less marked as the acute endo-pericarditis subsides, for physical signs are usually more pronounced during the acute stage, become less distinct and may indeed entirely disappear during the quiescent period.

The prognosis for this attack is excellent because the child has no fever now, and despite the murmurs, there are at present no cardiac symptoms and no decompensation. I wish to emphasize here the important distinction between physical signs and the actual cardiac condition of a patient. The former may be and indeed usually are of extreme diagnostic and prognostic importance, and of great scientific interest; yet, as in this case, they may furnish only an insufficient clue to the efficiency of the heart. Loud murmurs do not necessarily mean severe heart disease, nor do faint ones mean slight heart disease. In other words physical signs form only a part of the clinical picture and examination, and must always be correlated with other data, such as can be gathered from a careful history, from the patient's symptoms, and from the actual state of the circulation. For instance, if a patient be dyspnœic, or have edema, or an engorged liver, or cannot walk stairs without becoming inordinately short of breath, that patient has some severe cardiac lesion, whether typical murmurs be present or not. Regarding medication for this child, I shall advise salicylate of soda in five grain doses three times daily for about one week. She should be kept in bed for about two weeks; thereafter if there be no untoward symptoms, she may gradually begin to walk around, and perhaps at the end of a month, she may again go to school. It is necessary to give the child as much fresh air and sunlight as possible and to feed her up. It is of great importance to have her gain several pounds by proper dieting, for children thus fattened bear their disease much better than those of substandard weight, and the increase of weight is itself a good index that there has been general improvement.

### CASES 16, 17, 18.—RHEUMATIC AORTIC STENOSIS AND REGURGITATION; BOVINE HEARTS—REMARKS UPON GASTRO-INTESTINAL SYMPTOMS IN PATIENTS WITH AORTIC LESIONS

**Gentlemen.**—I present these three patients together because their histories and physical signs are similar, because their hearts are typical bovine or ox-hearts, and because in addition they have attacks of severe and widely distributed pains.

Case 16.—J. J., male, aged 32, states that his mother told him he had "palpitation" when five years old. Since the age of ten, he has had frequent precordial thumpings and pounding, especially upon exertion or when excited. He has had articular rheumatism frequently; the last time one year

ago. He often has attacks of what he considers indigestion, with eructation and belching. He also has attacks of chest pains and rapid heart action, which wake him up from sound sleep. In addition to these attacks, he has occasional spells of nocturnal pains, in which he has to "shake himself" for hours in order to get rid of the pain. He has had several attacks of constipation so severe that his family physician says they resemble intestinal obstruction.

**Examination.**—Upon feeling the radial, you note that the pulse is of the typical Corrigan type, the sudden collapse of the radial becomes more evident by raising the patient's arm above his head. The blood pressure reading corroborates the collapsing pulse; the systolic being 105; the diastolic, 0. You notice the tremendously throbbing carotids, the aorta in the jugulum and even the subclavians. You can see the immense precordial systolic heave which occupies the lower half of the left chest. Upon palpation, you readily feel the systolic thrill over the aorta in the second right interspace as well as in the jugulum, and the carotid thrill in the neck. You feel the jerky, diffuse ventricular impact almost as if the apical thrust were split up into several waves. Upon auscultation, you hear a very loud, rasping systolic murmur not only over the aorta, but also over the carotids and upper half of the right chest. The same murmur is transmitted posteriorly in the right and left interscapular regions. There is also a loud diastolic murmur, most intense over the right base, and transmitted with less intensity along the left border of the sternum. At the apex a soft systolic and a soft diastolic murmur are heard. There is no sensitiveness upon precordial pressure. The pulse is regular, the rate 80 per minute. The liver is not enlarged, there is no abdominal sensitiveness. There is no edema of the legs. The urine is normal.

Summarizing the physicial data, we find that the patient has an old rheumatic double aortic lesion, with tremendous left ventricular hypertrophy. The murmurs over the mitral area are probably referred from the base.

We shall reserve the discussion of the abdominal symptoms and the "shaking" attacks until we have completed a brief study of the succeeding two cases (Cases 17 and 18).

Case 17.—A. S., aged 26, male, married, says that he knows of no infection except measles, although he does recall that ten years ago he had pains in the legs lasting two weeks. His work consists in carrying heavy bundles. He smokes about ten cigarettes daily. The first cardiac symptoms consisted in occasional rapid heart action while at work. For four years he has had frequent peculiar night attacks. They would start with midsternal pains radiating to the back, accompanied by belching and sometimes vomiting. He would then have to get up and "stretch," and the pain would not be relieved until it passed "down the chest." The attacks lasted minutes or hours; they were usually followed by tachycardia. Neither diet nor exercise seemed to influence the onset nor the severity of the attacks.

**Examination.**—As in the previous case you note that this patient is tall and spare. The physical signs also are quite similar in both: The typical Corrigan pulse, the throbbing carotids and aorta, the immense precordial heave, the systolic thrill over the right base, aorta and over the carotids, the diastolic murmur heard over the right base and along the left sternal margin, the thrill-like jerky ventricular impact, the massive ventricular hypertrophy and the nocturnal attacks of pain. There is no precordial or epigastric tenderness; no edema of the legs. The urine is normal.

CASE 18.—I. L., aged 20, had articular rheumatism, with remissions, from his sixth to his eighth year. He has rarely had rheumatic attacks since then. He has had "heart trouble" for ten years. Until two years ago the symptoms consisted mainly in rapid heart action without dyspnœa. During the last two years, he has had peculiar attacks with increasing frequency. These are usually preceded by a day or two of anorexia and lassitude. The attack itself is usually nocturnal and is ushered in by a feeling of epigastric pressure which then radiates to the back, abdomen and legs. It is frequently accompanied by tachycardia. There is no vomiting. The pressure gives way to pain which becomes extremely intense, so much so that he literally writhes in agony as he actually did in one attack in which I saw him. Pressure over the abdomen seems to bring him some relief; for example, in the attack of which I speak, I found the patient's mother actually lying across his abdomen with her full weight. There is no precordial distress or vomiting during the spell. Mild attacks last several hours; severe ones, two to three days. It requires about a week for thorough recuperation and return to his old status.

**Examination.**—You observe how tall and lanky this boy is. The physical signs that you will find upon inspection, palpation, percussion and auscultation are quite similar to those of Cases 15 and 16.

It would be difficult to find a group of three patients with double rheumatic aortic lesions, in whom the physical findings were more similar or classical. The murmurs, the tremendous ventricular hypertrophy, the youth of the patients, the throbbing carotids and aorta, and the Corrigan pulse are the outstanding features.

It has always puzzled me to understand why the aortic valves become the site of such severe rheumatic disease in some young individuals. The patients we have just examined are of the thin, graceful type, with graceful rounded and slender extremities. Such individuals are regarded by some as belonging to the "lymphatic" type, with hypoplastic vessels and heart. If that be so, one can conceive that such substandard cardio-vascular systems may the more readily become the site of rheumatic infection. My own experience has been that the majority of severe rheumatic aortic cases in the male belong to this lymphatic type.

What caused the attacks in Cases 16, 17 and 18 with their accompanying phenomena? If rheumatic manifestations such as tonsillitis and articular pains had directly preceded the attacks, one might assume an added infective

insult to the heart as the primary agent. But such a history is absent. Rheumatic manifestations had not recently been present in any of the patients. From the history and course of the attacks, dietary indiscretions, excitement, and streptococcemia could also be excluded as etiological factors. What appears to me as the fundamental cause and the most likely one, is a continued progressive pathological change either in the cardiac muscle or in the endocardium. Where the damage is already as wide-spread as in these cases, and proper cardiac functioning is, even at its best, tremendously handicapped, increasing destructive changes need not necessarily be severe in order to still further compromise the circulatory mechanism. For example, interference with the coronary arteries by the hyperacting aorta or even their possible partial temporary occlusion by the massive weight of the heart might sufficiently alter the intracardiac circulation to evoke painful attacks. In addition, patients with marked aortic lesions are extremely susceptible to extraneous influences, such as excitement, as well as to pathological insults to the endocardium and myocardium. There apparently exists a hypersusceptibility of the nerve mechanism controlling the heart, brought about by constant excitation of the rich nerve supply surrounding the root of the aorta; this is caused by the tremendous hyperpulsatile activity of that vessel with consequent continued mechanical excitation of its surrounding nerves. Thus morbid impulses are sent to the corresponding dorsal segment of the spinal cord, and a vicious reflex arc is set up between the diseased aorta on the one hand, and a hyperexcitable spinal segment on the other.

It is such reasoning, admittedly theoretical in part, which I believe best explains the various phenomena exhibited by Cases 16, 17, and 18 during their attacks. For example, in Case 16 the intestinal symptoms were such as to sometimes resemble intestinal obstruction. Spastic constipation has often been ascribed to changes in the nerve control of the intestines; indeed, such patients have occasionally been operated on for intestinal obstruction although no mechanical cause has been found at operation. I believe the intestinal symptoms in Case 15 which always began with palpitation, constitute an instance in which the intestinal nerves became reflexly excited. In Case 17 the symptoms were those of esophago-spasm—sharp, midsternal pains radiating to the back, accompanied by belching—while in Case 18 the pains consisted of exceedingly severe intestinal colic. It is interesting to note that in all three, the patients attempted various mechanical procedures in order to relieve the pain: Shaking the body, the pressure on the abdomen, walking up and down.

### CASE 19.—BEGINNING RHEUMATIC DOUBLE AORTIC LESION—CONTINUANCE OF THE LESION DESPITE TONSILLECTOMY

C. W., male, aged 7, has had 'growing pains' for the last two years. The mother is positive that he has had only one attack of tonsillitis. One year ago both tonsils were removed. Soon thereafter he developed measles,

and a few weeks later, frequent attacks of rheumatic pain in the knees and feet. These attacks were accompanied by fever lasting several days, and by an itching rash somewhat resembling measles. The child has been kept in bed for his heart and rheumatism for several weeks at a time. It is important to emphasize that the child himself does not complain of a single cardiac symptom.

**Examination.**—This stocky youngster as you see does not impress one as being ill. His teeth are in good condition. Upon examining the pharynx, you observe that both tonsils have been radically removed. Upon inspection of the neck and chest, you notice the over-active aorta in the jugulum; there is also some cardiac hyperactivity noticeable in the apical region. Upon auscultation you hear a distinct systolic and diastolic murmur over the base of the heart, especially to the left. Both murmurs are transmitted slightly downward. At the apex, you hear a slight short presystolic murmur; this is probably the so-called Flint murmur. The remainder of the physical examination reveals nothing abnormal. The knee reflexes and the urine are normal. There is no edema of the legs. You find upon palpating the knees that both are somewhat tender, probably from a mild rheumatic process now present.

The orthodiascopic tracing being passed around shows a large and abnormally broad aorta. There is also moderate left ventricular hypertrophy. This is evident not only from the enlargement to the left but also from the contour of the left ventricle which is almost square in shape. It is evident that the boy is suffering from a double rheumatic aortic lesion. My main reason for presenting him is to have you observe the effect of the tonsillectomy. Just how large the tonsils were before operation we of course do not know. The history states definitely that the child has had but one attack of tonsillitis during his whole life, so that unless some marked, gross change in the tonsils had been present, I question the advisability of tonsillectomy after one attack of tonsillitis. You also recall from the history that soon after the tonsils were removed, the child suffered from rheumatic attacks. We know that such rheumatic attacks are apt to be accompanied by exacerbations of endocarditis. One may therefore speculate as to whether the operation did not do real harm instead of good. As I have already pointed out to you in discussing other cases, I feel that nowadays tonsillectomies are done much too often with the idea of controlling rheumatic infections and endocardial lesions. In cases which suffer frequently from tonsillitis or in those in whom the tonsils are particularly enlarged and look ragged and fleshy, tonsillectomy is no doubt indicated. But the time for doing the operation must be properly chosen, for if done during the active stages of endocarditis, or when rheumatic manifestations are still active, it may be followed by just such rheumatic and endocardial recrudences as we see in this boy. The operation should be done, if at all, when rheumatic and endocardial manifestations have ceased for some weeks or even months. The fact that practi-

cally every tonsil, when removed, shows some disease should cause no surprise, for lymphoid tissue situated at the portal of the pharynx must almost of necessity show some pathological change. Palpably diseased tonsils or those frequently inflamed should of course be removed, but as just pointed out, the time chosen for operation should be a properly chosen one, during a quiescent period of endocarditis if possible.

### CASE 20.—TYPICAL LUETIC AORTITIS

A. F., male aged 55, says that ten years ago he had gonorrhœa lasting several weeks. Some years ago, he was abed with "rheumatism," which lasted about a month. He had previously smoked as many as fifty cigarettes a day; at present, he smokes ten daily. For the last two years, he has had gradually increasing attacks of dyspnœa accompanied by precordial pains which radiate to the left arm. The attacks occur especially when he is aggravated, when walking in the cold air, or after sexual intercourse. He sleeps well at night. His appetite is good. He has not lost weight.

**Examination.**—You notice the patient is well preserved and is not dyspnœic when at rest. The radial arteries do not feel thickened, the pulsation is equal on both sides. Upon raising the arms above the head you note a tendency to the water-hammer pulse. The systolic blood pressure is 140; the diastolic is 40. Upon inspection you observe some overaction of the carotids. The aortic pulsation is not visible or palpable in the jugulum. Upon palpation there is no precordial sensitiveness, nor is there any abnormal pulsation over the mitral or aortic area. Upon auscultation over the right base you hear a typical double aortic murmur, a rough systolic and a loud diastolic, both of which are transmitted downward to the cardiac apex. Except for this transmitted murmur, the sounds at the apex are normal. There is some tenderness to pressure over the epigastrium. The edge of the liver is palpable slightly below the free border of the ribs. The pupillary light reflexes are sluggish. The knee jerks are normal. There is no edema of the legs. The urine is normal. The orthodiascopic tracing, as you readily observe, shows marked enlargement and broadening of the first part and arch of the aorta. The left ventricle is only slightly enlarged. The Wassermann blood reaction is 4 plus.

I have presented this case to you chiefly because the physical signs are so typical of aneurismal dilatation of the aorta. A double aortic murmur in a middle-aged individual is in itself suggestive of syphilitic aortitis. A rheumatic double aortic lesion without marked ventricular hypertrophy is rare in the middle-aged. This case illustrates a fact that I have often observed, namely, that extensive ventricular hypertrophy is by no means a necessary accompaniment of luetic aortitis; it may be present or the ventricles may be of normal size. On the other hand, the aorta is involved in almost every case of cardiac syphilis.

The therapy required in this patient is obvious. He should be put upon iodides and mercurial injections. He should also be given salvarsan injections, at first in small, later, in full doses. If the treatment be continued, the prognosis is good; the precordial pains and dyspnœa will probably disappear within several weeks. The aortic enlargement is permanent, but if further luetic inflammation be controlled and the disease be not far advanced, all the symptoms will probably recede.

### CASE 21.—SYPHILITIC MYOCARDITIS—CARIES OF TEETH—GINGIVITIS—PYORRHŒA—REMARKS UPON THE CONNECTION BETWEEN HEART DISEASE AND PYORRHŒA

**Gentlemen.**—This man is forty-seven years old. He has been married eighteen years, his wife has never been pregnant. He had gonorrhœa many years ago; he denies syphilis. He had double pneumonia at one time. Five years ago he had "rheumatism" in the muscles of the shoulder and forearm. For the last seven years, there has been sugar in his urine in amounts up to 1 per cent.; it is always readily controlled by proper diet. He had trouble with his teeth for many years; at various times he has been treated for pyorrhœa. All of the teeth of the lower jaw have been extracted; there is considerable bridge work in the upper. Despite local treatment by good dentists, pyorrhœa is still present. Cardiac symptoms began about two years ago. These consisted chiefly in a feeling of oppression on the chest, of dyspnœa, and of frequent expectoration of blood-stained sputum. He has also had several attacks of sudden dyspnœa with expectoration of frothy sputum. He has had epigastric pain and tenderness for several months; these symptoms were ascribed to indigestion by his family physician.

**Examination.**—As the patient removes his set of lower false teeth, you note that the gums of the lower and upper jaw are spongy; they bleed readily, pus can be squeezed out of the upper tooth sockets. The patient is slightly dyspnœic when at rest, the respiration rate is 25 per minute. He looks well nourished. Inspection of the precordium reveals nothing abnormal. Upon palpation we note there is slight overaction in the apical region, there are no abnormal pulsations over the right base or in the jugulum. Superficial or deep pressure over the heart elicits no pain or sensitiveness. Auscultation reveals a normal first sound over the right base, the second aortic is somewhat metallic in character. At the apex, with the patient sitting up, the first and second sounds are distant and indistinct; when the patient lies down, you note the increased dyspnœa; the cardiac rate rises to 100 per minute. When the patient is in the recumbent position, you hear at the apex, typical "gallop rhythm," a generic, and, I believe, an unscientific name given to any reduplicated sound. In our case I think the gallop rhythm is due to a reduplication of the first sound, a matter of some importance in establishing the diagnosis of myocarditis. The cardiac area is somewhat enlarged to percussion. The systolic blood pressure is 130; the diastolic, 60. The lower

border of the liver is palpable two inches below the free border of the ribs. There is distinct epigastric tenderness to pressure. The knee reflexes are normal. There is no edema of the legs. The urine is normal. There are a few scattered rales at the bases of both lungs. The radials at the wrist are not thickened.

You observe that in this case there are but few physical signs upon which to base even a generic diagnosis of heart disease, let alone of myocarditis. We must therefore depend chiefly upon the symptoms and the history. The prominent symptom is dyspnœa, especially upon walking. There is nothing in the lung examination, urine or blood pressure which sufficiently accounts for the dyspnœa. The frequent hemoptyses, and the attacks of extreme dyspnœa and frothy expectoration no doubt indicate edema of the lungs of varying severity, and point to the heart as their cause.

What are the physical findings which substantiate myocarditis? As already stated, the physical findings are merely suggestive, not definite. They consist in slightly rapid heart action, especially with the patient in a recumbent position; in distant heart sounds and in the reduplicated first sound at the apex (so-called gallop rhythm). These physical signs are in themselves not sufficient evidence upon which to base a diagnosis of myocarditis, but they must be studied in conjunction with the history and symptoms. I consider the so-called "indigestion" and the epigastric sensitiveness valuable evidence, not of gastric but of heart disease. I have frequently found such signs in other frank cases of cardio-vascular disease. Indeed I am always suspicious of *cardiac*, not of gastric disease, if, when coupled with dyspnœa a patient complains of "indigestion," or has epigastric pains when walking as well as on abdominal palpation. Such pains are commonly ascribed to pressure of the stomach against the diaphragm and the heart. My roentgenographic studies of such cardiac cases with opaque barium meals have led me to believe that the pains are not caused by such assumed pressure of the diaphragm against the heart, for I have often found the stomach small and hypertonic, containing only small, normal amounts of air; hence undue pressure against the diaphragm could scarcely be a factor in disturbing the circulation. Although I possess no definite proof, my clinical observations incline me strongly to the belief that most cases of epigastric pain with heart disease are instances of referred and reflex phenomena having their origin in the heart.

I am usually loath to call a case myocarditis unless there are other unmistakable evidences of severe cardio-vascular disease: Such as frank decompensation, hypertension, cardiac hypertrophy, aortitis, etc., but I believe the symptoms and history of the present case are sufficient for the diagnosis of myocarditis, without other evidence.

**Etiology.**—Here again this patient is of great interest, because the only apparent cause for the disease lies in the condition of the gums and teeth. I belong perhaps to the small minority who feel that the role of the teeth as the

cause of heart disease has been tremendously overestimated. I have seen heart disease as frequently in those with, as in those without decayed teeth and pyorrhœa. It is one thing to find all sorts of pathogenic and pyogenic bacteria in the gums and carious teeth, and another, to prove that these have entered the blood stream or have produced toxins and thus cardiac damage. On the other hand, occasionally one cannot deny the possible etiological connection between a pus focus in the mouth and heart disease. I believe, however, that in every instance, connection between heart disease and carious teeth must be definitely proven, before a patient can be said to have heart disease from bad teeth. The case before us seems to offer no other cause for the myocarditis than the foul condition of his mouth. His "rheumatism" several years ago may well have come from the same cause. I shall, however, have a routine Wassermann blood test made to exclude syphilis as a possible etiological factor.

**Report and Examination Two Months Later.**—You recall, gentlemen, this case of myocarditis that I demonstrated to you several weeks ago. You recall also that I believed this a rare instance in which pyorrhœa was the probable etiological factor of heart disease. Two days after you saw this man, I had a Wassermann blood test done; the report stated that it was 4 plus. I immediately put the patient upon vigorous anti-luetic treatment—iodide of potash in increasing doses, intra-muscular injections of salicylate of mercury and salvarsan intravenously. He has had three doses of the latter, the first dose was two decigrams and the others were six decigrams each. He no longer has any dyspnœa, he can now walk rapidly, as you see, without shortness of breath, the blood-tinged morning expectoration has ceased; even his gums and pyorrhœa are much improved in spite of frequent mercurial injections. The physical signs of the heart are about the same, except that the heart is not as rapid, and you hear the reduplicated first sound only after exercise. The case, therefore, gentlemen, is one of syphilitic myocarditis—the positive Wassermann and improvement under anti-luetic treatment have proven that. Despite the history and the condition of this man's mouth, our protean friend, syphilis, is after all the cause of this patient's heart disease.

### CASE 22.—AORTIC ANEURISM AND AURICULAR FIBRILLATION

M. F., male, aged 47, a hard-working individual, had never complained of his heart until three months ago. He then had "palpitation." The doctor who saw him at that time says that the patient had an attack of edema of the lungs. Since then he has been unable to work because of tachycardia, dyspnœa and epigastric distress when walking. He denies any gonorrhœal, luetic, rheumatic or tonsillar infection. The only previous illness he remembers is a slow healing ulcer of the leg following a trauma some years ago.

**Examination.**—You observe that the patient is slightly short of breath even when at rest. On looking very carefully with the eye on the level of the chest, you note a slight pulsatile rise in the second and third right intercostal

spaces. There is no pain on precordial pressure. In the apical region you feel the irregular ventricular activity—strong and weak, fast and slow beats following each other with no regularity. The pulse is also quite irregular. This irregular pulse and heart action is typical of auricular fibrillation. Upon auscultation you hear a soft systolic murmur over the second, third and fourth right interspaces; but I want you especially to hear the resonant, twanglike, accentuated second sound over this same area. There is no diastolic murmur over the right base. The sounds over the mitral area are normal. The systolic blood-pressure of most of the effective beats is around 190; the diastolic, around 130. The liver is not palpable. There is sensitiveness to pressure in the epigastrium. There is no edema of the legs. The urine is normal.

How shall we interpret the physical signs? Let us attempt to do so even before we fluoroscope the patient. I believe that the pulsatile rise of the tissues in the right intercostal spaces, combined with an accentuated second sound over the same area is indicative of aneurismal enlargement of the first portion of the aorta; the enlarged aorta acts here somewhat as a resonator or sounding board in producing the accentuation.

Fluoroscopy discloses as you see, much more enlargement than I had suspected. There is not alone aneurismal dilatation of the first portion of the aorta, but there is a large sacculated aneurism of the first portion, arch and descending thoracic aorta. The left ventricle is only moderately enlarged. The process is undoubtedly luetic. We shall get a Wassermann report of the blood in a day or so.

How shall we explain the fact that the patient had been able to work for years and then was suddenly attacked with pulmonary edema and subsequent decompensation? I believe that the actual cardiac complaints date from the onset of fibrillation, for to judge from the size of the aneurism, the lesion must have been a steadily progressive one. Without a more exact previous history than we have been able to obtain, it is impossible to state the cause of fibrillation. However one may well imagine the final involvement of some vital cardiac structure such as the coronary arteries, as the culminating cardiac "insult" which produced the arrhythmia. I do not believe that there is sufficient myocarditis present to cause auricular fibrillation. The X-ray does not show massive ventricular enlargement; and progressive myocarditis would probably have been accompanied by signs or symptoms of decompensation long ere this.

Dating probably from the occurrence of fibrillation, we have the sudden change of a man with a very large aneurism without cardiac complaints to one who is now dangerously ill. It is necessary to emphasize again a proper understanding of the importance of *slow* progression of an organic disease. Thus this aneurism was undoubtedly of many years steady growth. But it had not caused any undue pressure upon surrounding structures, and, to judge from the absence of cardiac symptoms, there has been no inflammatory

exacerbations; the only handicap the heart and circulation had suffered was the purely physical one of sacculation of the root and arch of the aorta.

You recall the fluoroscopic picture of only moderate ventricular enlargement. In my experience, I believe that the degree and constancy of ventricular hypertrophy which accompanies aortic aneurism has been very much overestimated. Indeed, I have examined patients with large aneurisms in whom there was no appreciable ventricular enlargement. The usual impression of huge hypertrophy is doubtless gained by the exaggerated ventricular thrust against the chest wall during systole, and by the low and forward position of the ventricles which are commonly crowded downward and to the left by large aneurisms.

**Later Report.**—The Wassermann report came back 2 plus. The patient was placed upon large doses of the tincture of digitalis. Salvarsan injections and mixed treatment were to have been instituted by the family physician in a few days. Before proper treatment could be carried out the patient had a sudden severe attack of epigastric pain, vomited and died within a few minutes. Coupled with the previous history, the cause of death seemed due to a coronary infarct.

### CASE 23.—LUETIC AORTITIS—CONSTANT PRECORDIAL PAIN

I present this case in contrast to Case 22 because constant and long continued pain is the main symptom.

H. P., male, aged 44, complains of very severe, almost constant substernal pain for two years. When pain is especially severe, it radiates to the left arm and back and is accompanied by dyspnœa. The attacks are occasionally relieved by nitrite of amyl. In addition the patient often finds it necessary to take ¼ of a grain of morphine sulphate every night. The last Wassermann reaction was 4 plus. He has had six neo-salvarsan injections with no effect upon the symptoms or upon the cardiac status. The urine contains a trace of albumen but no casts.

**Examination.**—You readily see, even from a distance, the throbbing carotid and brachial arteries. The systolic and diastolic pressures of the right and left brachials are respectively, 170–20, and 150–40. The patient is now having one of his usual attacks of pain. We shall immediately give him five minims of nitrite of amyl contained in a pearl, and study its effect upon the blood pressure. The systolic blood pressure in the right arm has fallen to 100. This fall is but momentary, lasting as you note, just one minute; none the less, the inhalation seems to have afforded the patient some relief, for he says that he feels better and that his attack is over. Upon inspection of the chest you observe the pulsatile rise of the aorta in the jugulum, and the tremendous ventricular hyperactivity. These observations are readily confirmed upon palpation. Upon auscultation you hear a rough, rasping first sound over the right base and in the jugulum, and a markedly accentuated and resonant second sound over the right base. The orthodiascopic tracing

being passed around shows aneurismal dilatation of the first part and arch of the aorta, and extreme left ventricular hypertrophy. The electrocardiogram shows normal rhythm with evidence of left ventricular preponderance—a negative deviation in the third lead. The liver is palpable but not tender. There is no edema of the legs.

In contrast to the previous case (Case 22) this patient has been suffering from almost continuous precordial pain for two years. He uses as many as one dozen 5 minim nitrite of amyl pearls daily, and although he fights against the use of morphine as much as possible, he finds it necessary to take one dose at night time, otherwise the pain becomes unbearable.

The diagnosis is of course plain: The patient has an aortic aneurism and marked left ventricular hypertrophy. The direct source of the pains seems to be due to involvement of the coronaries; this etiology appears more probable by the relief the patient finds in amyl nitrite.

The outlook for the cure of the precordial pains seems remote. Thorough anti-luetic treatment has brought the patient no amelioration. This probably means that the cardiac tissue, especially the coronaries, have reached such a stage of sclerosis that they can no longer be influenced by anti-luetic therapy. However I shall advise proper digitalization of the patient in the hope, rather than the expectation of helping the intra-cardiac circulation. Mixed bromides in large doses, and tablets containing nitro-glycerin and atropine sulphate will also be advised.

**Later Report.**—The family physician reported to me that the patient was not benefited by the medication and is still clinging to amyl nitrite and morphine.

### CASES 24, 25, 26.—SENILE CARDIO-SCLEROSIS—LOUD SYSTOLIC MURMUR OVER THE ENTIRE ANTERIOR SURFACE OF THE CHEST—GENERAL COMMENT

I shall first present to you a brief history of each of the three cases, after which we shall discuss in detail the peculiar and interesting physical findings of all.

Case 24.—C. L., male, aged 68, suffered from moderate dyspnœa at the time of hospital admission. The systolic blood pressure is 130: The diastolic, 100. The urine contains a trace of albumen. The cardiac rhythm is normal; the rate between 76 and 80 per minute. Over the entire middle anterior aspect of the chest, you will hear a loud, rough systolic murmur transmitted slightly to the left axilla and along the carotids. The roentgenogram reveals an aortic arch almost twice the normal width, and a somewhat enlarged left ventricle.

Case 25.—M. C., female, aged 64, complained of cough and dyspnœa. Six months prior to hospital admission she suffered from some precordial distress, the exact nature of which could not be determined. The temperature was 102°; there was moderate dyspnœa; and there were physical signs

of general bronchitis. The fever and bronchitis lasted two weeks. Upon examination, you will find that the cardiac area is somewhat enlarged to percussion; a loud, rasping, systolic murmur is heard over the entire precordium; it is especially intense over the right base. The murmur is transmitted along the carotids and laterally to the left axilla. It is also heard as a muffled systolic bruit posteriorly in the upper interscapular region. The systolic blood pressure is 220; the diastolic, 140. The urine contains a moderate amount of albumin and some hyaline casts. The roentgenogram reveals not only slight dilatation of the aortic arch but also aneurismal dilatation of the entire descending thoracic aorta. The latter as you see is a prominent shadow on the left side of the chest from the second to the fourth left interspace; at that point it is lost behind the shadow of the left ventricle.

CASE 32.—J. M., male, aged 66, complains of epigastric pain, dizziness, headache and some dyspnœa. There are old scars, possibly syphilitic, on the forearm. The lungs appear normal. Over the entire precordium you hear a loud, rough systolic murmur. The cardiac area is somewhat enlarged to percussion. A roentgenogram reveals moderate enlargement of the aortic arch. The cardiac rhythm is normal. The systolic blood pressure is 135; the diastolic, 95. The urine contains a slight trace of albumin.

I am grouping these cases because, as you observe, they present the same auscultatory phenomenon, namely, a loud, rough, systolic murmur which is heard over the entire anterior surface of the chest.

Perhaps I had better define the conception—cardio-sclerosis—because it is apt to be loosely used. When I use this term I refer to marked pathological changes involving the cardiac muscle, the valves, the aorta, and the coronaries and their branches. The damage is by no means equally distributed among these structures; frequently the aorta and the coronary system are chiefly affected.

To return to our patients. You observe they all possess the usual characteristics of generalized arterio-sclerosis; thickened, palpable and tortuous arteries. Pulsation of the aortic arch in the jugulum is a prominent visible and palpable physical sign in all.

The systolic murmur—the special reason for presenting these three cases—is rough and loud, and is heard over the entire front of the chest. Its areas of greatest intensity are over the right base and the lower precordium. It is also transmitted along the carotids and the lower left axilla. In Case 25 it is heard posteriorly as a soft, muffled sound in the upper interscapular region.

After but a casual examination, one may be inclined to ascribe the murmur to the presence of a large sacculated aneurism or to aneurismal dilatation of various parts of the aorta. Both types of aortic enlargement, however, are usually accompanied by diastolic, as well as systolic murmurs, the area of greatest intensity being over the right base of the heart. In aneurismal dilatation of the descending thoracic aorta—a rare, isolated condition—one

usually finds the double murmur loudest in the third left interspace near the sternum; from this location, it tails away toward the apex and base. This characteristic is absent in Case 31. In cardio-sclerosis, the loud systolic murmur is heard with or without aortic enlargement; the diastolic murmur is absent. In addition, careful auscultation reveals a marked difference in the intensity of the murmur over the precordium. It is very loud over the right base, losing in intensity over the midsternum, again becoming louder at the cardiac apex and to the left. It thus seems as if the murmur is made of two components—an aortic and a mitral. The former is probably caused by such arterio-sclerotic changes as deformities and thickening of the wall of the aorta. The mitral component of the murmur may be ascribed to the characteristic degenerative changes affecting the mitral valve and the mural endocardium of the mitral ring, with consequent production of a typical mitral regurgitant murmur. The aortic and mitral components are both systolic in time; hence, when the pathologic changes are of the gross nature above outlined, a loud systolic murmur results, which may be of fairly equal intensity over the entire front half of the chest, or there may be an area of less distinctness and intensity over the midsternum.

### CASES 27, 28.—CARDIO-SCLEROSIS IN THE MIDDLE-AGED—LOUD SYSTOLIC MURMUR OVER THE ENTIRE SURFACE OF THE CHEST

Case 27.—C. H., female, aged 50, buxom in appearance, has had three children and no miscarriages. For years she suffered from severe rheumatic (?) neuralgic headaches. For one year she had precordial oppression and attacks of severe precordial pain radiating to the jaw, back and left arm. A rest cure lasting several weeks brought no relief. The blood Wassermann is negative.

**Examination.**—The systolic blood pressure is 170; the diastolic, 110. The report of the blood chemistry states that the non-protein nitrogen and other constituents of the blood are normal in amount. You notice that there is no dyspnœa when the patient is at rest. On palpation, slight overaction of the carotids and of the aortic arch is evident. Over the right base, you hear a loud, rough systolic murmur transmitted along the carotids; it is also heard posteriorly in the mid-dorsal region to the right of the spine. Below, anteriorly, it becomes less intense and is merged with an extremely loud systolic murmur, most prominent and distinct over the mitral area. The orthodiascopic tracing reveals an enlarged aortic arch and a somewhat globular and enlarged left ventricle.

Case 28.—A. B., a physician, aged 54, of vigorous appearance, contracted nephritis following a grippe infection about twenty years previously. Cardiac complaints began about four years ago. They at first consisted of occasional hemoptysis and of slight dyspnœa. Of late months the dyspnœa has increased and is accompanied by a sense of weight on the chest. The Wassermann blood reaction is negative.

**Examination.**—You observe that the apex beat is diffuse. Over the entire anterior aspect of the chest you hear a loud, muscial systolic murmur, most intense over the right base and the lower precordium; it is least intense over the midsternum. It is transmitted along the carotids, and is also heard posteriorly at the third dorsal vertebra. The orthodiascopic tracing reveals an enlarged aorta as you see, and a hypertrophied left ventricle.

I am briefly demonstrating these two middle-aged patients in the same group with the three patients of the senile cardio-sclerotic group (Cases 24, 25, 26) for in both the physical signs are the same, except that the thickening and tortuosity of the radials and temporals are absent in these middle-aged cases. Indeed, to judge merely from the facies, both patients, C. H. and A. B., appear in robust health and give no inkling of the profound cardiac disease we find upon examination. Their comparative youth speaks for the fact that the pathological inroads upon the heart took place at a relatively rapid rate, thus apparently causing more serious and progressive loss of the normal cardiac functions.[1]

### CASE 29.—CARDIO-SCLEROSIS—REMARKS ON THE CAUSE OF SYMPTOMS IN SENILE CARDIO-SCLEROSIS

Mrs. E., aged 73, gives the following history. She has had five children, no miscarriages. She had pneumonia twelve years ago. She does not recall any other acute illness. While a young woman she suffered from attacks of diarrhœa for two years. She finally developed gastric symptoms: Belching, eructations, and a dry, irritating cough; the latter was often accompanied by belching. Since five years, she has what she describes as a cold feeling in the left chest anteriorly. This feeling is often relieved by belching. Several weeks ago, she had a severe hemorrhage following the extraction of a tooth. She has always been active in business, attending to her work as a designer for years. Five weeks ago, immediately after moving a heavy piece of furniture, she experienced a sudden sharp pain in the left breast. Since then she has a "faint," sore feeling in the left chest, is dyspnœic and has occasional "palpitation." The precordial sensations are often relieved by belching.

**Examination.**—You observe, gentlemen, that this elderly patient is well preserved, with the keen and active intelligence of a much younger person. Yet even superficial inspection shows marked evidence of general arterio-sclerosis, as well as of cardio-sclerosis. The temporal and radial arteries are visibly and palpably thickened. Inspection reveals overacting carotids, and a pulsatile heave of the tissues in the jugulum from the overacting aorta. As I raise the left breast, you observe the diffuse precordial heave due to the enlarged heart. The apex beat is markedly displaced, being near the axillary line in the sixth interspace. Upon palpation the ventricular impulse is felt as a diffuse overforceful shock. There is a small area in the left precordium

[1] Both patients died of edema of the lungs within a few months of the time they were demonstrated.

that is sensitive to moderate pressure. Upon auscultation over the right base the first sound is rough, the second, bell-like. Over the apex there is a systolic murmur accompanying the first sound. There is slight edema of the legs. The liver is palpable two inches below the free border of the ribs. The blood Wassermann is negative. The urine contains a slight trace of albumin, a few granular casts, and no sugar. The systolic blood pressure is 180; the diastolic, 70. The orthodiascopic tracing being passed around shows aneurismal enlargement of the first portion of the aorta and marked left ventricular hypertrophy.

This patient, like several others we have studied, has typical generalized arterio-sclerosis and cardio-sclerosis with massive ventricular hypertrophy. It must have taken many years for these pathological changes to have occurred. Two main questions are of interest to us in this case: (1) Why was this patient free from cardiac symptoms for so many years? (2) What caused the sudden onset of the symptoms?

Regarding the first question, it is often puzzling to understand why patients with apparently similar lesions suffer from such diverse symptoms. I have shown you other patients with extreme cardio-sclerotic changes who have suffered from severe cardiac symptoms and decompensation for years. What appears to me of prime importance in the production of cardiac symptoms in cardio-sclerotics, is the *rate of progress* of the disease. One can readily conceive that *rapid* destruction will quickly overwhelm the function of an organ and will soon cripple it, whether that organ be the heart, liver, kidneys, or any other. But when, for any reason the pathological process attacking the cardio-vascular system is slow in its progress, and no vital part is attacked, the function may be so gradually interfered with, that the normal reserve power of the heart—its "factor of safety"—is only very gradually weakened. Such a view, of course, considers the heart chiefly from the standpoint of function. This is oft-times advisable, for when the mind, eye, and ear are too engrossed in minute pathology, we are prone to forget the importance of the validity and power of the still remaining healthy portion of an organ. For instance, I have little doubt that our patient has marked coronary changes. The aortic changes, the physical signs and the generalized arterio-sclerosis indicate the likelihood of such coronary damage; yet a gradually progressive coronary change may only slowly cut off the vascular supply of the cardiac musculature. It may thus give time for the beginning of a collateral circulation, for we no longer regard the coronaries as end-arteries. Sudden infarction in coronaries not nearly as diseased as we assume those of this patient to be, may end in sudden death. We thus see how it is possible for some patients with severe cardio-sclerosis to live fairly comfortably despite their severely damaged hearts. You also see that even minute physical examination does not tell us everything about a patient, and that at times broader view-points must be considered in order to understand a case from all angles.

With reference to the second question, "What caused the sudden onset of the symptoms?" The history clearly states that the pushing of a heavy piece of furniture suddenly brought on the cardiac symptoms: Pain, dyspnœa and palpitation. It is probable that any great effort in a person with a severely damaged heart makes sudden demands upon the circulation by quickly raising the blood pressure and by increasing the force and rapidity of ventricular action, thus initiating cardiac symptoms. I have observed a similar case in a much milder instance of cardio-sclerosis; precordial pain followed the lifting of a heavy bundle. Such conditions cannot be better characterized than by calling them by the somewhat unscientific and generic term, cardiac strain. The amount of strain required to produce cardiac symptoms will naturally vary with the amount of circulatory reserve; this in itself is a variable factor, and is rarely determinable in advance of the supreme circulatory effort that finally produces cardiac symptoms from overstrain.

I believe that for the present this patient should be given as much physical rest as possible. This does not necessarily mean in bed, for some old people become restive and worried when in bed; they feel that their last days have come, and that they will never be about again. Such views may seem old-fashioned to you, but they are very real to the patients, so that unless definitely indicated, it is unwise to put older patients to bed. They may lie upon sofas, or rest in easy reclining chairs. This patient should be given intensive digitalis therapy for one week: About a drachm and a half of the tincture of digitalis for four days, followed for three days by theobromine sodium salicylate and a restricted fluid intake. If dyspnœa, edema and precordial pain be benefited, the chances for the re-establishment of excellent compensation are good, and it may even be possible for the patient to again take up, at least partially, her old employment of designer.

**Later Report.**—The patient remained at home several weeks. The dyspnœa soon ceased and the edema disappeared after two weeks. The precordial sensitiveness has almost entirely gone. The patient at first was allowed to walk about the room, then upon the street, and finally permitted to go to business for a few hours each day. The family physician reports that now, several months from the time we first saw her, she is again at business, quite as active as formerly and rarely complains of any of her heart symptoms.

### CASE 30.—CARDIO-SCLEROSIS—CHOLECYSTITIS

M. J., female, aged 64, seven years ago had an attack of right-sided abdominal pain of three months' duration. There was no jaundice. Several years thereafter, she developed midsternal pains, especially when walking in the open against the wind; the pains usually radiated to the right shoulder and right breast. Three months ago, she developed gastric symptoms; belching after meals, and rather constant epigastric distress aggravated by eating. She has lost 25 pounds in weight during the last year. She has

been sent here to determine whether the epigastric pains are of gastric or cardiac origin.

**Examination.**—The systolic blood pressure is 160; the diastolic, 80. The urine is normal. Inspection of the chest reveals as you notice nothing abnormal. Upon palpation, there is a small area in the midsternum sensitive to pressure. Over the right base and over the mitral region, you hear a loud, rough systolic murmur, most prominent when the patient is lying down. The orthodiascopic tracing shows a somewhat enlarged aorta; the other cardiac outlines are normal in form and size. There is no edema of the legs. Abdominal examination reveals tenderness over the gall bladder region, with the pain radiating to the right side of the back.

I do not wish to enter into the details of the abdominal examination, except to state that the latter is very suggestive of cholecystitis. The loud murmur and the hypertension present definite evidence of cardio-sclerosis. And yet, as you see, there is no decompensation: The patient is not dyspnœic and she can walk comfortably about the room. I believe that the cardiac nerves of this individual have become hypersensitive because of the abdominal condition, whether of cholecystitic or of gastric origin. In other words the reflex cardiac arc has become abnormally excited and has thus been rendered hyperirritable. That is to say, the midsternal pains are not necessarily indicative of severe coronary disease, but are symptomatic of a hypersensitive reflex arc, and hence the morbid reaction—pain—even in the presence of *mild* coronary disease.

The cardiac condition presents no contraindication to operation if such be indicated because of the abdominal complaint. There is no decompensation, nor are the cardiac symptoms suggestive of any acute inflammatory disturbance.

### CASE 31.—ALCOHOLIC CARDIO-SCLEROSIS

J. K., aged 63, married, has three grown up, healthy children. He considered himself well until two years ago. He then had what the doctor told him was an attack of edema of the lungs. About that time dyspnœa began. At first it was nocturnal in character; later it was also present by day. Of late, with exacerbations and slight remissions, dyspnœa has been a constant feature. He also has a dry cough. He denies any venereal history. Until his fortieth year, he drank an average of three or four glasses of red wine daily; he had never been intoxicated.

I have had this patient under observation for one year. His condition when I first examined him was quite different from that at present. There was then definite evidence, on palpation and percussion, of considerable left ventricular hypertrophy. There was a loud systolic murmur over the mitral area and at the second right interspace. The cardiac rate was 110 per minute with occasional extrasystoles. There were signs of fluid at the right base posteriorly. The liver was very much enlarged. There was slight

edema of the legs. The urine contained a slight trace of albumin and a few casts. The blood pressure was then 140 systolic and 70, diastolic. A Wassermann blood test was negative. A nitrogen partition test of blood showed that the non-protein nitrogen, creatinin and uric acid were normal in amount. The phthalein output for two hours was also normal.

The patient was put upon intensive digitalis and theobromin sodium salicylate therapy. The tincture of digitalis was given in twenty-drop doses, three times daily for one week, followed for three days by theobromine sodium salicylate in one-half gramme doses four times daily. The fluid intake, chiefly water and weak tea, was restricted to about 500 c.c. (one pint) in twenty-four hours. At the end of several weeks, there was marked improvement in the patient's condition. The dyspnœa almost entirely disappeared; the liver diminished in size; edema of the legs also disappeared and the patient was able to walk about comfortably.

This favorable status continued several months. Gradually, however, the condition in which you now find him supervened. You note his apathy, his somewhat cyanotic lips and the brief attacks of dyspnœa, during which the entire facies becomes more cyanotic. The cardiac examination reveals, even upon cursory inspection, the diffuse precordial heave betokening massive ventricular hypertrophy. Upon auscultation the murmurs I described as being plainly heard a year ago are now scarcely audible; this is probably due to weakness of the heart action. The regular rhythm is frequently interrupted by what appear to be extrasystoles occurring in groups of three or four. There is massive edema of the lower extremities. The urine contains a slight trace of albumin and some granular casts.

To sum up—this patient presents a definite clinical picture of cardio-sclerosis. I believe the predominant pathological change is in the heart muscle, the patient has marked myocarditis. While the etiological factor is not absolutely certain, I think that the habitual drinking of wine in earlier manhood is responsible for the present cardiac status. Syphilis can be excluded, not only because of the negative Wassermann, but also because he has not responded to mixed treatment given him before coming under my observation. About a generation ago, alcoholism was held largely responsible for many cases of heart disease. According to the modern conception, the pendulum has swung very far in the opposite direction, and alcoholism is now regarded as a very rare cause of cardiac damage. Both views are extreme I believe. Each case must be studied on its merits; the habits of the patient must be very carefully investigated, and other factors of course, must be carefully excluded. From this patient's history and by a process of exclusion, I believe that he is suffering from alcoholic myocarditis.

Why is this patient so listless? He is apparently not uremic, but just as his legs are edematous, so also is the brain probably water-logged and edematous. Such a condition naturally interferes with proper cerebral functioning.

It is of interest to inquire why medication—digitalis and theobromin—at first so successful in relieving the decompensation, is at present of no avail. It must be remembered that myocardial degeneration of such marked degree can never be entirely restored to its normal state. Drugs can only restore, to some extent, the driving force, the pumping power of the heart, and the degree to which it attains this success must depend upon the amount of healthy muscle susceptible to drug action. This presupposes that no sudden cardiac accident, like a coronary infarct, has supervened to alter entirely the pathological picture. The underlying condition, alcoholic myocarditis, is a slowly progressive one, and when degeneration is already extreme, drugs can scarcely stay or halt the increasing pathological damage. This patient will grow progressively worse, and, unless some other accident such as edema of the lungs occurs, he will die with the typical picture of heart failure.

### CASES 32, 33.—MYOCARDITIS WITHOUT HYPERTENSION OR PHYSICAL SIGNS

CASE 32.—M. W., male, aged 64, had always led a quiet and regular life and was not given to excesses of any kind. He is married. His only previous cardiac complaints were occasional left sided precordial pains and dyspnœa when taking long walks. Neither was sufficiently severe to cause him to seek medical advice. One week ago, while going to business, he felt nauseated and later vomited. He was dazed but managed to arrive home alone. At home he complained of left sided pains in the chest radiating into the left arm. He had no cough. The pains gradually subsided but he had several severe attacks of dyspnœa. There was no fever. Two days after the attack he developed edema of both legs. His physician states that the urine then contained a trace of albumin but no casts.

**Examination.**—You note that the patient's face is somewhat gray. He is not dyspnœic at this moment although the doctor who attended him states that he was suddenly called to see him after midnight yesterday because of extreme shortness of breath. The dyspnœa must have been intense, for the patient himself states that he spent the preceding night in a rocker because he could not get his breath. The systolic blood pressure is now 120; the diastolic, 85. The urine is normal. Inspection and palpation of the precordium reveal nothing abnormal. There is no parasthesia or sensitiveness to superficial or deep pressure over the heart. Upon percussion the heart seems somewhat enlarged to the left. The first sound at the apex is faint and distant; when the patient lies down this sound is reduplicated (so-called gallop rhythm). You hear a faint systolic murmur which accompanies the first sound at the apex. You will find that the murmur becomes somewhat louder when the patient is in a sitting position. At the base of the heart the sounds are normal. Abdominal examination reveals nothing abnormal. Upon auscultation of the lungs you hear some mucous rales at both bases. The radial arteries do not feel thickened. You see that there is edema of both lower extremities, chiefly of the right leg.

**Re-examination 24 Hours Later.**—You now find that the cardiac status of the patient is about the same. Despite vigorous stimulation, edema of the legs is still present. The patient was uncomfortable and dyspnœic during the night. The rales in both lungs are more numerous and widespread. The patient's mentality is now somewhat dulled. These signs presage an early death from heart failure.[1]

In this patient I venture the diagnosis of myocarditis for the following reasons: The history points to a coronary infarct on the day the patient first became ill: The nausea, vomiting, precordial pains and dyspnœa were doubtless due to this cause. A coronary infarct also satisfactorily accounts for the resultant cardiac failure—edema of the legs, and, later, pulmonary edema. Assuming the diagnosis of coronary infarct as correct, it seems highly improbable from the pathological standpoint that coronary disease can be present as an isolated lesion unaccompanied by more or less widespread disease of the cardiac musculature. It is upon this assumption that I base the diagnosis of myocarditis even in the absence of other criteria of the disease. The absence of auscultatory evidence of myocarditis will be discussed in conjunction with the following case.

Case 33.—E. S., aged 53, male, was said to have had "heart trouble" when he was a soldier in Switzerland at the age of 20. His family physician who has had him under careful observation for 10 years, states that he has had slight shortness of breath and occasional moderate tachycardia for a number of years. In that interval his systolic blood pressure varied from 100 to 143. The urine was always normal. The patient never presented any cardiac murmurs. He never had any edema of the legs. The only previous illnesses of note are an old gonorrhea in his youth and a severe attack of facial erysipelas 20 years ago.

During the past few months the patient has become increasingly dyspnœic and has developed substernal pains. He has great difficulty in climbing the hill leading to his home and must often stop because of shortness of breath and pains in the chest. Two days ago his physician states that he had a typical attack of pulmonary edema. This was relieved by the hypodermic administration of morphine sulphate and of digitalis.

**Examination.**—As you look at this patient now, he is the picture of health. His complexion is ruddy; he has no discomfort in breathing. There are no abnormal jugular or carotid pulsations. You hear at the apex a slightly reduplicated first sound, more marked when the patient sits up. Otherwise the cardiac examination reveals nothing abnormal. There are no rales to be heard in the chest. There is no edema of the legs. The systolic blood pressure is low, it is 80; the diastolic, 60.

I present these two patients chiefly to demonstrate that both have severe cardiac disease—probably coronary disease and myocarditis; yet the most careful physical examination elicits no abnormal signs except the gallop

[1] The patient died two days later.

rhythm at the apex. These cases illustrate the importance of a minute study of such symptoms as dyspnœa and precordial pain, and of the history of attacks of edema of the lungs. These of themselves offer sufficient evidence of severe heart disease without the usual physical phenomena to be found upon cardiac examination. In the first patient (Case 32), the beginning symptoms were gastric, and the pains in the chest and arm might have been ascribed at the outset to a stomach derangement. But increasing dyspnœa and edema of the legs soon cleared up the clinical picture and made it apparent that the heart was the offending organ.

### CASE 34.—PREGNANCY WITH CARDIAC DISEASE AND ENDOCARDITIC RECRUDESCENCE

Mrs. W., is now in the fourth month of her fourth pregnancy. The other pregnancies and deliveries were normal. The youngest child is ten years old. Although she knew she had "heart disease," she had no symptoms until four years ago. At that time she had slight hemoptysis, probably from a pulmonary infarct. Since then she has had difficulty in walking because of pains in the epigastrium and under the left breast. These symptoms have been aggravated during the last six months; they are accompanied by "palpitation." During the last few months, she has had severe palpitation during coitus. Since her pregnancy, she has had several attacks of cough, fever, dyspnœa and blood-streaked sputum.

**Examination.**—You see the patient is somewhat dyspnœic especially when attempting to lie flat. Over the mitral area you hear the classical murmurs of a double mitral lesion; these are also heard distinctly posteriorly between the vertebral column and the angle of the left scapula. The second pulmonic sound is accentuated. The cardiac area is not enlarged to percussion. You hear distinct signs of localized dry pericarditis over a small area at the third left interspace near the left sternal border. The heart is regular; the rate 100 per minute. The temperature is 102° per rectum. Over the lungs posteriorly, near the scapular angles, you hear localized sibilant breathing and mucus roles; over the right side there is in addition distant broncho-vesicular breathing. There is epigastric tenderness to pressure. The liver is not enlarged. The blood pressure and urine are normal. There is no edema of the legs.

Since the attack of hemoptysis the patient has not been able to walk without pains or palpitation. During her pregnancy these have become aggravated. Lung complications have been added—attacks of bronchopneumonia with fever. Dry pericarditis is now present; whether this is a recent or old process it is impossible to state. This case exactly fits in with my experience that when pregnancy occurs during the active recrudescent phases of endocarditis, it has the effect of aggravating existent symptoms and of adding new complications. There are two factors which must always be considered

in conjunction with pregnancy and heart disease: Decompensation and endocarditic recrudescences. I believe the latter is the more dangerous and leads to more fatalities than mild decompensation. Gestation in some unknown manner seems to light up dormant or only partially active endocarditic processes.

I shall advise this patient to have an abortion done at once. I believe it unsafe for her to attempt to carry her pregnancy to term.[1]

### CASES 35, 36.—PREGNANCY WITH CARDIAC DISEASE AND NEUROTIC SYMPTOMS

CASE 35.—Mrs. K., aged 21, married one year, is pregnant three months. She has had "heart disease" for years. In the past ten years her only complaint has been occasional attacks of rapid heart action. Since her pregnancy she has had frequent crying spells because obsessed with the idea that she would die during childbirth. She complained almost constantly of palpitation.

**Examination.**—You see a vigorous woman who is not dyspnœic. Upon auscultation you hear the typical signs of a double aortic lesion: A rough rasping systolic and a softer diastolic murmur over the right base; the latter is transmitted to the left and below. There is moderate ventricular hypertrophy. The cardiac rhythm is regular, the rate being between 100 and 110 per minute. Compensation, you observe, is perfect—there is no dependent edema and no dyspnœa, despite the tachycardia.

I shall comment upon this case after the examination of the next patient.

CASE 36.—Mrs. L. is married two years. She is now pregnant six weeks. She has had "heart disease" since childhood but has always been able to play and romp with other children. Since her tenth year she has never been in bed because of her heart. She is of a neurotic temperament and is readily frightened. It is when frightened that she suffers from momentary tachycardia.

**Examination.**—Upon exposing the chest you note even at a distance the exaggerated ventricular action. The cardiac rate is 110 per minute and is regular. Typical signs of a double mitral lesion are present: A rough diastolic rumble and a loud systolic murmur over the mitral area. There is no evidence of decompensation. The patient confesses that for nights she does not sleep because she dreads that she will not live through her pregnancy and childbirth. Her heart is almost always "jumping."

What advice should be given to these young women? Should the pregnancy be continued or interrupted? Both have perfectly compensated lesions; Case 35 has a double aortic, Case 36, a double mitral lesion. In both the lesion has been quiescent for many years. Because of the knowledge

[1] The next day, the gynecologist gave the patient a small injection of scopolamine, and dilated and packed the uterus. After several days, the fetus was expelled. Thereafter, all signs of bronchitis and bronchopneumonia disappeared; further convalescence was uninterrupted; palpitation and precordial pain entirely disappeared.

of their cardiac disease both have become frightened and obsessed by the fear of death from childbirth. The result has been quite similar in both; crying spells and tachycardia. It is really the tachycardia per se which requires symptomatic relief. Without the latter symptoms, I should have unquestionably advised the continuation of pregnancy, for the two grave dangers in pregnant women with cardiac disease—decompensation and rheumatic endocarditic exacerbations—are here absent. I should consider even this patient with the double mitral lesion (Case 36) a safe case for the continuation of the pregnancy if tachycardia were absent. I say this, despite the fact that patients with mitral stenosis are not usually considered good risks for pregnancy and childbirth. I believe, however, that by careful study and discrimination, one may select cases in whom pregnancy and childbirth may be advised. For example I have advised the continuation of pregnancy in several cases of mitral stenosis with no untoward results: Childbirth and puerperium were normal. Besides, we occasionally observe patients with mitral stenotic lesions who have gone through several normal pregnancies and who never knew that they had heart disease.

To return to our patients—they are so extremely nervous and fretful that I am confident that neither reassurance, medication, nor rest will allay or control, even in part, their basic dread of pregnancy and death.

Since both are in the very early stages of gestation I would advise immediate surgical intervention for the termination of pregnancy. In this stage and with compensated patients, an abortion, skillfully and aseptically performed, adds no operative risks to the cardiac lesion. I shall, however, explain to both these patients, that it was their nervousness, rather than the actual condition of their hearts which called for operative interference, and that if at some future time their hearts remained as good, and if they were less frightened, they would have no "palpitation," and pregnancy would very probably proceed normally.[1] A good general rule is that pregnancy should not be allowed to continue in those who have shown evidence of decompensation or of fresh endocarditic attacks within two years of conception.

### CASE 37.—OVERACTING HEART—ABNORMAL PHYSICAL SIGNS—REMARKS UPON A RATIONAL METHOD OF OUTLINING THE HEART

J. H. B., male, aged 14, has grown very much during the last year or two. He joins in all sports. His only complaint is occasional left-sided abdominal pain after running. There is no history of rheumatism or tonsillitis. The boy is supposed to have "heart disease."

**Examination.**—As you see, this patient is quite tall, fairly muscular and of the blond type. The teeth and tonsils are normal. The systolic blood pressure is 130; the diastolic, 87. Upon inspection of the chest you see sil-

[1] The family physician recently reported to me that Case 35 had had an abortion performed. Within a few months, she again became pregnant, and had a normal delivery and puerperium. The patient herself is feeling well.

houetted quite plainly through the rather thin chest wall, the overacting heart, especially at the apical region. Upon palpation you feel a thrill-like, slapping apical impulse especially when the cardiac rate is increased by exercise. Upon auscultation you hear a thrill-like, split or broken first sound at the apex; it is systolic in time and is not accompanied by any murmur. The apical thrill is unchanged when the boy lies upon his back. Over the second right interspace there is a soft systolic murmur which diminishes in intensity with inspiration.

I now invite your attention to the percussion findings. Following the ordinary method of percussion, you observe that there is increased dullness to the left, less to the right. This therefore presupposes cardiac enlargement to the left. The orthodiascopic tracing, however, which I am now showing you, reveals an organ that is quite normal in contour and size, and corresponds with the size of the heart one would expect to find in a boy of his physique. The urine and knee reflexes are normal. There is no edema of the legs.

There are two points of special interest in this case: The abnormal physical signs upon auscultation and palpation, and the discrepancy between the percussion and orthodiascopic findings.

A thrill-like first sound heard upon auscultation and felt upon palpation is by no means infrequent in overacting normal hearts. Its cause is not quite clear to me. In some instances, it may be caused by intraventricular eddies and swirls so to speak, resulting from abnormally violent heart action; at any rate by itself it has no pathological significance and is not indicative of any myocardial or endocardial lesion.

The soft cardio-respiratory murmur at the second left interspace is extremely common, especially in the young.

With reference to the marked discrepancy between the orthodiascopic and percussion outlines this is but another example of what careful comparative studies have long since proven to me; namely that percussion findings are very inexact and often valueless as compared with X-ray examinations. Orthodiascopy itself undoubtedly gives exact outlines because it deals with parallel rays. I have tried many methods of percussion: Ortho-percussion, mediate, immediate, stroke, light and heavy—and I have never been able to detect any correlation between the cardiac outlines thus studied and the actual size of the heart as found by the X-ray. A moment's thought on the physics and acoustics of cardiac percussion will perhaps explain the reason for this discrepancy. The heart is a muscular, more or less soft organ, situated at different depths from a chest wall of varying thickness. The great vessels —the aorta and pulmonary arteries—with their contained blood are of softer consistency than the muscular mass of the heart. An added complicating factor is the amount of lung tissue—an air-containing, light substance—between the heart and chest. Because of the difference in densities of the cardiac structures themselves, and because of the variations in the thickness of the

wall and the intervening lung pad, it seems *à priore* highly improbable that any percussion method, or any combination of methods can give even approximate data of the size of the heart and aorta. I have therefore attempted to apply the lessons I have learned from fluoroscopy to percussion. I reverse, somewhat, the usual routine employed in physical examination. I seek first the most prominent visible and palpable apical impulse by inspection and palpation. If this point is not sufficiently visible, I auscultate to define it more accurately. From fluoroscopy I have learned that the actual apex may be from 2 to 6 c.m. below this point. In thick-set individuals there is comparatively slight mobility of the diaphragm, while in those with graceful chests, diaphragmatic excursion is marked. Hence in the former, the actual apex is near the point of maximum impulse, while in the latter it is much lower. I have corroborated these facts by frequent fluoroscopic examinations. The upper aortic line is next delimited. The finger is placed in the jugulum behind the manubrium. If aortic pulsation can be felt, a corresponding line is marked on the manubrium. Auscultation, and the diagnosis of the cardiac lesion, if there be any, will determine how far the aortic pulsation in the jugulum may be regarded as being pathological. If such pathological lesion in the aorta cause an undue, overpulsatile rise, the result will be an abnormally high level for the palpated aorta. The approximate position of the aortic valves is next determined by auscultation over the right base. The area at which the aortic sounds are loudest marks fairly accurately the position of these valves; the upper limit of the right aortic curve is about 2 c.m. above this point. The usual palpation and auscultation is then practiced. These with the history and the remainder of the clinical findings determine the absence or presence of cardiac disease. If heart disease is absent, the remainder of the cardiac outline (between the aortic curve and the apex) can be determined and blocked in by a knowledge of the probable shape of the heart in the particular type of chest wall. (See Orthodiascopy, Chapter XII.) If a cardiac lesion has been diagnosed, the remainder of the cardiac contour can also be fairly accurately foretold with the aid of palpation and other clinical data; it is then drawn on the chest wall by our orthodiascopic knowledge of corresponding types of heart disease, with and without decompensation.

I admit that this method has its drawbacks for it is founded partly on inferences. However, I have not made any of the gross errors inherent in percussion, and while often wrong I have not found wide or wild discrepancies between the cardiac outline as determined by my method, and the actual orthodiascopic size of the heart.

I have made this digression because I wanted to justify the fact that in previous clinics, I passed over the subject of percussion very cursorily. Let us now return to our patient.

An important factor is that a rapidly beating or overacting heart always gives the impression of being larger than it actually is. Such is the case

here. The orthodiascopic outlines are normal, although the result of the examination by auscultation and palpation gives the impression of considerable cardiac enlargement.

The physical signs—the thrill-like apical sound heard and felt with cardiac systole—are not the result of any cardiac disease in our patient, but simple come from cardiac hyperactivity. Other cases that I shall show you will also demonstrate abnormal physical signs without organic cardiac disease.

### CASES 38-43.—IRRITABLE HEART WITHOUT ORGANIC HEART DISEASE—REMARKS COMPARING THE IRRITABLE HEART IN CIVIL PRACTICE WITH THAT OF SOLDIERS

CASE 38.—J. H., male, aged 27, unmarried, says that in his business as a grocer he had occasional slight discomfort in lifting heavy boxes. This never interfered with his business until five years ago; he then had an attack of severe abdominal cramps. He felt faint and his heart action became very rapid. The doctor who saw him then and with whom I have communicated, stated that his heart action was regular and the rate 180 per minute. Since that attack, nervousness, excitement, attempts at hard work, or full and hearty meals are accompanied by rapid heart action and precordial pains. His appetite is good; he has not lost weight. He is nervous and worried about his heart, more so since he has been drafted and accepted for war service.

**Examination.**—The patient is quite robust and well nourished. His thyroid is not palpable. The systolic blood pressure is 110; the diastolic, 90. Inspection of the chest reveals slight apical overaction. You note upon palpation in the apical region, a systolic, almost thrill-like shock. There is no pain or sensitiveness to superficial or deep pressure. Upon auscultation you hear a systolic broken first sound, giving the impression of a thrill. The other heart sounds are normal. The cardiac area is normal to percussion. The urine is normal. The abdominal examination reveals nothing abnormal. There is no edema of the legs. The knee reflexes are somewhat exaggerated. When at rest the pulse rate is 100 per minute; the rhythm, normal. After moderate exercise as for example, walking a dozen paces rapidly, the pulse rate is 150 per minute and the patient complains of dyspnœa and some precordial distress; after about three minutes of rest, the pulse returns to its previous level.

This case is typical of irritability and instability of the nerve control of the heart. The history definitely indicates that an attack of paroxysmal tachycardia from some gastric disturbance five years ago initiated the cardiac symptoms. Prior to that, the patient was a hard-working individual, his only complaint being the not uncommon one of a sense of precordial oppression from heavy lifting. Now, excitement or effort is very apt to bring on rapid heart action, not of the paroxysmal variety with extreme acceleration, but with rates reaching 150 per minute, and accompanied by dyspnœa and

precordial pain. This tendency has been exaggerated by his dread of active war service. Despite the physical signs on palpation and auscultation, I believe organic disease can be excluded. I have found identical signs—a thrill-like systolic shock on palpation and a thrill-like first sound on auscultation—in a number of recruits of draft age in whom, after exercise, cardiac action was unduly accelerated or overforceful. The abnormal signs just mentioned are not evidence of organic disease in such individuals, for often when the cardiac rate or activity can be controlled (for example, by having the patient take and hold a deep breath) these abnormalities disappear. There is nothing in the patient's history—no antecedent rheumatic or other infection—which can be correlated with organic cardio-vascular disease. That is very important from the etiological standpoint, for it helps us to distinguish murmurs and thrills due to accelerated and violent action per se, from similar signs due to actual heart disease. In other words, we are dealing with a condition of hyperirritability and instability of the neurogenic control of the heart, originally induced by an attack of paroxysmal tachycardia.

We shall discuss the general problems of the therapy and etiology of the irritable heart later, in conjunction with remarks upon the soldier's heart.

Case 39.—J. T., aged 28, a furnace worker, has been married five months. His work is very laborious. He has had slight precordial pains, and pains radiating into the left arm for some months. The symptoms were aggravated after a dispute with a fellow-laborer some three weeks ago. He has lost thirteen pounds in weight during the present illness. He has had occasional palpitation and dyspnœa.

**Examination.**—You note the strong musculature of the thorax and arms, indicative of continued hard labor. The lungs are normal. Inspection shows slight carotid overaction. There is no pain on palpation; the apical impulse is marked. Upon auscultation there is a slight thrill-like systolic first sound at the apex. The other cardiac sounds are normal. Fluoroscopic examination shows that the heart is normal in size and shape; overaction of the pulmonary artery and aorta is also evident. The thyroid gland is not enlarged. The abdominal examination reveals nothing abnormal. The systolic blood pressure is 142; the diastolic, 72, The urine is normal. The knee reflexes are very lively. The pulse rate varies suddenly from the normal to 100 per minute. This acceleration occurs with and without effort. The acceleration can be considerably, although temporarily, moderated by having the patient take and hold a deep breath. I always regard this phenomenon as an evidence of good vagus control.

This patient presents no evidence of organic disease. A dispute with a fellow laborer has apparently aggravated a dormant cardiac neurosis. I believe the man should give up his work for a week or two, not because the work itself would damage his heart, but because his present state of hypersusceptibility requires a rest. Bromides in moderate doses for a few days will help in the same direction.

Case 40.—H. M., aged 26, single, gives a questionable history of rheumatism in childhood. He had always been well and athletic until five years ago when he witnessed the drowning of his brother. He then became hysterical and was confined to bed two weeks with hysterical attacks. For two or three years thereafter, he had "nervous indigestion," the main symptom being belching. The gastric condition improved. The patient then developed cardiac symptoms—"palpitation" and pains in the left chest, particularly after excitement. After walking a mile now, he becomes "tired all over." He has recently been told that his heart is occasionally irregular. He is a very rapid eater. His present business—theatrical manager—keeps him so occupied that he has no time for relaxation.

**Examination.**—The systolic blood pressure is 120; the diastolic, 60. The thyroid is not enlarged. You note upon inspection slight carotid overactivity, and upon palpation slight exaggeration of the apical impulse. The sounds over the base are normal. At the apex, immediately after a normal first sound, you hear a sharp, superficial click, which is transmitted to the axilla; it disappears upon deep inspiration. Occasionally, a superficial pleuro-pericardial rub is also heard in this region. There is no precordial sensitiveness. The pulse rate is normal. Rapid walking around the room, as you see, produces no dyspnœa. The orthodiascopic tracing which I took of him shows that the cardiac outlines are normal in size and contour; the ventricular contractions were particularly powerful. There was no roentgenographic evidence of pericordial adhesions. The abdomen is not sensitive to pressure. The urine is normal. There is no edema of the legs.

I believe the adventitious sound at the apex is due to an old pleuro-pericarditis, and has no relation to the cardiac symptoms now present; it is simply an accidental finding. There is no other sign of cardio-vascular disease. The whole demeanor and action of the patient during the examination, as you probably observed, was that of an individual who is hyperactive; it cannot be better expressed than by the use of the common, though unscientific expression, "his nerves are tense; he is always on edge." Indeed, the history elicits the fact that he never acts calmly, that he has no time for actual rest, that his mind is always busily looking ahead. Whether his entire disposition was changed by having witnessed his brother's drowning, or whether the latter incident only intensified an inherent tendency, we cannot state because we possess no details antedating the accident. The history however leaves no doubt that the original shock, followed by hysteria, was the immediate starting point for a train of nervous symptoms, first affecting the stomach, and later, the heart. I want to emphasize again the fact that, although the cardiac rate was normal, and was not unduly accelerated after exercise, the "palpitation," although subjective, was actual to the patient, and caused the various cardiac symptoms of which he complained. Another point of interest is the question, would this patient make a good soldier? Since there is no tachycardia, the fundamental question is his attitude toward

the war. If he is imbued with the proper patriotic spirit to take a manly part, I believe that camp routine, with its associations and release from the hurried and harried life this man has been leading, will cause a reversion to a physiological, normal level, that the cardiac symptoms will disappear, and that he will only suffer from the usual fatigues incident to the work of the soldier.

CASE 41.—L.R., aged 48, married, salesman, has always led a very active business life but states that he has always been of a nervous temperament. He had "palpitation" when about 20. He has always been fond of exercise and sports. He frequently suffers from cold hands and feet. Since several months, he has had disputes with his wife because of business reverses. He is afraid to exercise now because he believes he has heart trouble. He never has any dyspnœa while at his work. His family physician tells me that he has had occasional extrasystoles and that an occasional hyaline cast is found in the urine. At present the chief symptoms are "palpitation" especially at night, and attacks of perspiration with clammy hands and feet.

**Examination.**—You see that the patient is of athletic build. The systolic blood pressure is 118; the diastolic, 95. If you feel his hands and feet now, you will find them cold and wet despite the warm temperature of the room. The pulse is regular, its rate, normal. All the cardiac sounds are normal. Rapid walking does not cause any abnormal increase of the heart rate. The orthodiascopic tracing now being passed around shows that the aorta is normal in size and shape, and that the remainder of the cardiac outline is somewhat broader than normal. The knee reflexes are lively.

I believe that organic heart disease can be excluded. The patient presented no arrhythmia during our examination, but the "palpitation" at night, and the family physician's statement that he had detected extrasystoles, make it probable that the night attacks are due to extrasystoles. The vasomotor instability—perspiration and clammy extremities—is the chief feature of the case. I do not regard the presence of an occasional cast in the urine as indicative of renal disease, for such a finding is common enough in perfectly healthy and normal individuals.

The first therapeutic measure I would suggest is to allow the patient to go on with his business, to assure him that his heart is sound, and then gradually to let him take up his usual athletics again. Of equal importance is the avoidance of bickerings at home; unless that source of irritation be eliminated, all other therapy will be of no avail. For a time and to hasten improvement, the patient should take 30 grains of mixed bromides at night, and occasional doses of the solid extract of the suprarenal gland when the vasomotor or cardiac symptoms become especially annoying.

**Later Report.**—The patient has told me that the directions given were faithfully carried out, and that he felt so well after two or three days that he is again not only attending to business but is taking up his usual athletics.

I feel convinced that assurance of the absence of heart disease was the controlling factor in this rapid cure.

CASE 42.—Mrs. W., aged 52, has had five children and several miscarriages. The menopause occurred two years ago; at that time she had occasional slight flushes for two months. Ten years ago she had gall stone attacks for which she was treated in Carlsbad; but she has had no recurrence in the last few years. Her husband died suddenly in bed two years ago. She was very much upset by his unexpected death. Since then she began to have palpitation, especially at night, and dreaded going to bed for fear that she, too, might die suddenly. She also had palpitation when excited or when walking in the street; this was accompanied by sharp substernal pain which occasionally radiated to the left arm. The systolic blood pressure, taken at different times, varied between 180 and 200. She was told of this hypertension and also told that she had arterio-sclerosis. She has been on a rigorous diet, and although often hungry (at which times she would experience faintness and tachycardia) she was afraid to eat for fear of increasing her blood pressure.

**Examination.**—The patient you see, is a stout woman, she is not dyspnœic when at rest. The thyroid gland is not enlarged. The systolic blood pressure is 150, the diastolic, 100. There is overaction of the carotids and aorta. There is no pain on superficial or deep precordial palpation. There is a strong systolic impact felt at the apex. Upon auscultation you observe that all cardiac sounds are normal; there are no murmurs nor accentuated sounds. When the patient is at rest, the cardiac rate is 110; it is slightly decreased upon deep quiet inspiration. Walking causes a rapidity of 120; after a few minutes it drops to 110. There is no edema of the legs. The urine and knee reflexes are normal. The orthodiascopic tracing which I here demonstrate to you shows a heart of normal size and form.

In spite of the history of hypertension and of precordial attacks, I regard the case as one of pure cardiac neurosis due to fright, and reflexly aggravated by hunger pangs. The latter were probably caused by a starvation diet which kept the stomach empty a good part of the time, and induced gastric hypermotility. I shall allow the patient a liberal mixed diet, to be eaten at regular intervals. I shall prescribe bromides at night and two grain tablets of the extract of suprarenal gland for the attacks of tachycardia.

**Later Report.**—Within three weeks her condition improved remarkably. The systolic blood pressure was constantly around 130, the precordial pains disappeared and the cardiac rate fell to 80 per minute. The patient sleeps well at night and can walk regularly in the open without discomfort. Reassurance doubtless was a large factor in her improvement.

CASE 43.—Mrs. G., aged 26, has been married one year. She has a baby three months old. Seven years ago she had a love affair; she became disappointed in her lover and gave him up. She then had many hysterical spells, finally culminating in attacks of severe nocturnal dyspnœa. Since then she

has had many such attacks. She finally married another suitor and became pregnant. While pregnant the attacks ceased entirely, but they have recently recurred. Her home life is now a happy and comfortable one. During a typical attack her hands and feet become cold and the heart action, rapid. She becomes dyspnœic, there is a feeling of palpitation and also of abdominal pressure; accompanying these are intestinal hypermotility and rumbling. The patient says she tries to control the attacks, which usually come on and are severest at night. She is losing weight and is afraid to go out because of the attacks. Her appetite is poor.

**Examination.**—The teeth and tonsils are normal; the thyroid not enlarged. Now the patient states that she has what she considers a mild attack. Her hands, you observe are cold and clammy. Inspection of the cardiac area is normal. There is a broken, thrill-like systolic shock palpable, and a thrill-like sound heard over the mitral area. The other heart sounds are normal. The cardiac rate is 110 per minute; walking about in this room has increased it to 120; it fell to its previous level within five minutes. The abdomen presents nothing abnormal. The knee reflexes are very lively. There is no edema of the legs. The orthodiascopic tracing shows a heart normal in size and shape.

I consider this case a typical example of an irritable heart made so originally from the emotional strain of a love affair years ago. The symptoms subsided during pregnancy, apparently because the pregnant state caused a profound change in her mental attitude, a common result of pregnancy. A hyperfunction of one or more of the endocrine glands may have been the fundamental cause of such change. After childbirth, there was recurrence of the old symptoms, chiefly vasomotor instability and dyspnœa. Although originally hysterical, I do not consider the present attacks of that nature; rapid heart action alone, as you know, can cause dyspnœa. The presence of symptoms due to irritability of the vasomotor center seems to indicate in addition, a possibility of hyperexcitability of the respiratory center. Studied with a respiratory apparatus, such patients are sometimes found to be suffering from tachynpœa (simply rapid breathing) rather than from dyspnœa.

My views of cardiac neuroses with full consideration of the etiological factors involved and of the abnormal physical signs and phenomena, as well as the therapy are given in detail in Chapter XIV and XVIII; hence they require no repetition here.

### CASES 44, 45.—FAINTING ATTACKS FROM SO-CALLED WEAK HEART—REMARKS ON "WEAK" HEART

Case 44.—A. L., married, aged 49, fainted five years ago. He then had no gastric symptoms. From that time and until the attack of four days ago, he felt well. While sitting at a banquet in a room full of smoke, he felt weak and lost consciousness. Both fainting attacks were attributed to a "weak" heart.

**Examination.**—This patient you observe is a vigorous man. The systolic blood pressure is 138; the diastolic, 72. The urine is normal. You find that all the heart sounds are normal. Now that he has walked rapidly around the room you also find that the heart action is not inordinately rapid nor violent, nor does the patient become dyspnœic. The knee reflexes are normal. There is no edema of the legs. The orthodiascopic tracing being passed around shows a normal-sized aorta; the ventricular area is somewhat broader than normal, probably because the ventricles are pushed up by the diaphragm, a frequent finding in stout individuals; this therefore gives a false impression of ventricular enlargement.

I find no evidence of cardio-vascular disease or of "weak heart" in this individual. The fainting was probably caused by cerebral vaso-motor changes from breathing impure air in a warm and stuffy room. The patient should be allowed to again follow his usual occupation. He requires no treatment.

Case 45.—S. K., unmarried, aged 27, states that he has suffered from fainting spells for years. The sight of blood causes him to faint. These attacks are most apt to occur when he is in a warm room. Sometimes there is a sense of light-headedness instead of an actual faint. The attacks have no relation to diet or exercise; indeed, he is very athletic; plays tennis and rides horseback. He is a very moderate smoker. He has no gastric or intestinal disturbance. The family physician states that at times the pulse is irregular, and that the heart sounds are faint.

**Examination.**—You see this patient is an excellent example of an athlete; tall and lithe. The teeth and throat are normal. Upon auscultation you find all the heart sounds quite normal, without any evidence of "weakness." When the patient lies down, the heart sounds become somewhat louder. The pulse and cardiac rhythm are occasionally disturbed by a premature contraction (an extrasystole), of which the patient himself is not aware. Inspection and palpation of the precordium reveal no abnormality. The blood pressure and urine are normal. There is no edema of the legs. The orthodiascopic tracing being passed around shows a normal sized aorta, and a large sized heart, but not exceptionally so for an individual of his physique.

I shall now have you fluoroscope the patient with me. Watch the ventricular contractions carefully and you observe, not "weak" ventricular systoles, but just the reverse. You note the vigorous ventricular contractions, as shown by the decided diminution in the size of the ventricle with each systole.

Cardio-vascular disease can be excluded, I believe, as a cause of fainting spells and of the extrasystoles; both are probably due to vaso-motor instability. I have found that the extract of suprarenal gland is of value in these individuals, if it can be given at the first premonition of faintness or light-headedness. This patient may continue his business and follow his usual exercises, no matter how vigorous or even violent they may be.

Both cases typify what is popularly termed "weak heart," yet neither has the slighest evidence of cardiac disease, and in the case we examined fluoroscopically, you were actually able to see the strong cardiac contractions. The importance of vaso-motor instability from whatever cause —impure air, gastric disturbances, fright, etc.— as a cause of fainting is very frequently overlooked; the heart is accused as the offending organ, and patients are sometimes coddled to almost ridiculous degrees because heart disease is mistakenly regarded as the source of the trouble. This is especially true when some cardiac irregularity—usually extrasystoles—co-exists. Of course, the entire cardio-vascular apparatus must be carefully examined in these individuals, so as to positively exclude any organic lesion as the cause of fainting attacks. The treatment does not consist in rest and quiet, nor in giving digitalis. Treatment must be directed to the cause of the vaso-motor instability. As indicated, these are manifold. In every case, therefore, it is necessary to search for the special cause and to treat it appropriately.

### CASE 46.—VASOMOTOR INSTABILITY—REMARKS ON SO-CALLED "WEAK HEART"

Mr. L., aged 60, always worked hard. He has never complained of his heart. Ten years ago he had a long-continued attack of diarrhœa which he attributed to milk. At that time he complained of occasional "thumping in his chest." Since that diarrhœal attack and until a month ago his bowels have been regular, but there is usually slight abdominal colic immediately preceding every evacuation. A month ago, he had another severe attack of diarrhœa from some unknown cause. Since then he has had frequent "weak spells" which consist of sweats, of cold extremities, and of a feeling of faintness. The spells last about one-half hour. They are occasionally accompanied by coliky pains and are sometimes relieved by the passing of flatus. The patient has been in bed for several weeks because he had been told that he had a "weak heart."

**Examination.**—The patient is well nourished; there is no dyspnœa even when lying flat. The radial arteries are not thickened. The pulse is regular; its rate, 60 per minute. The blood pressure is normal. There is no abnormal carotid or aortic pulsation. In fact, you will find that the most careful physical examination of the heart fails to reveal anything abnormal except a very soft systolic murmur at the apex. There is no abdominal tenderness, no edema of the legs. The urine and knee reflexes are normal. The orthodiascopic tracing, a copy of which you see, shows no enlargement of the aorta nor of the remainder of the cardiac outline.

The patient's symptoms I believe are real, not hysterical; an otherwise hard-working man is not apt to want to be bedridden. The diagnosis of "weak heart," made by another physician, has undoubtedly frightened him. I find no pathological cardio-vascular basis for the symptoms. The heart is normal. Of course such a statement can be made only after a most careful

examination, for you know the frequency of cardio-sclerosis at this patient's age. I do not consider the faint apical systolic murmur of any significance; it is such a common occurrence that in itself it is no criterion of cardiac disease. This patient is suffering primarily from disturbance and instability of the vaso-motor mechanism, a symptom-complex frequently attributed to so-called "weak heart." That term is a palpable misnomer, for the heart is really not "weak," there is never any actual decompensation in these individuals. The vasomotor mechanism is very susceptible to reflex influences, and in this patient it was probably reflexly excited by the diarrhœa which began several weeks ago.

It is of prime importance, therapeutically, to assure the patient that there is nothing wrong with his heart. He should be given tablets, each containing $\frac{1}{150}$ of a grain of atropine sulphate, three times daily before meals. After meals, one half a teaspoonful of an ant-acid powder containing equal parts of magnesia usta, bicarbonate of soda and oleosaccharated peppermint should be administered; depending upon its action on the bowels, the dose of the powder may be increased or diminished. For the vaso-motor attacks themselves, I know of nothing better than the extract of suprarenal gland given in tablets of two grains each. The diet must be carefully regulated, for anything which produces indigestion is very apt to aggravate the vaso-motor symptoms. It is therefore advisable to ask the patient what simple nourishment best agrees with him, and to have him follow such dietary; but the meals should be small, and taken at frequent intervals. Aggravation and excitement readily upset these individuals, especially their vaso-motor system, hence mental quietude and relaxation must be insisted upon. I shall keep the patient in bed for a few days longer, because if allowed on his feet after his long stay in bed, the vaso-motor instability is apt to increase, and the symptoms made worse. Although the patient is not ill it requires full and detailed knowledge of the cause of the symptoms in order to re-establish normal vaso-motor equilibrium. Attention to small and apparently trifling details is the sure road to success in these cases. I have known of individuals in whom insignificant domestic excitement has caused a relapse for several weeks.

**Later Examination.**—You saw this patient a few weeks ago. He remained abed for a week after you saw him; he then was allowed to walk around. He is now able to walk rapidly, and in a day or so will return to his work. His recovery has been complete and uninterrupted.

### CASES 47, 48.—IRRITABLE HEART FROM PHYSICAL OVERSTRAIN

CASE 47.—A. B., physician, aged 32, has had no scarlet fever, genito-urinary or other infection except an attack of rheumatism lasting one week; this occurred nine years ago. He states that in former years, he had occasional extrasystoles the cause of which he did not know. About six months ago, after riding horse-back for many hours, he suffered from severe precordial pains and extrasystoles. The pains were very acute and severe

for several hours. They later subsided although still aggravated by walking. They gradually disappeared entirely until about three months ago. The patient then took a Turkish bath; shortly thereafter, he suffered a recurrence of the precordial pains upon walking fast in the open air and upon climbing stairs. Otherwise he feels well in every particular although he still has an occasional extrasystole.

**Physical Examination.**—As you see, the doctor's teeth and tonsils are normal. You will find by careful inspection, that there is some hyperactivity of the aorta in the jugulum, and of the left ventricle against the chest wall. You can corroborate this by placing one finger in jugulum, and the palm of the other hand over the apical region. The cardiac rate is now normal, there is no irregularity of rhythm at present. The heart sounds are normal. Upon exercise there is no abnormal increase of the cardiac rate. The abdominal examination reveals nothing abnormal. The knee and pupillary reflexes are normal. There is no edema of the legs. The urine is normal. The orthodiascopic tracing being passed around shows a somewhat large heart. It is interesting to note that the fluoroscopic examination showed particularly strong ventricular contractions. The electrocardiogram shows normal rhythm; the deviations are practically normal in size and form. The therapy and symptoms will be discussed in conjunction with the following case.

Case 48.—S. J., aged 24, says that she had scarlet fever and diphtheria as a child and that she has suffered for years from severe hemicrania. She does not know the cause of the latter. She has never suffered from gastric disturbances. She does her own housework. Her menses are regular. She has one child. One year ago, after a heavy day's work, she dragged her child on a sled up and down hill for quite some time. The same evening she developed rapid heart action which has since continued. It is aggravated by overwork, climbing stairs and by excitement. Her appetite is fair, although she has lost ten pounds since her trouble began. Her bowels are regular. She has an almost constant feeling of dyspnœa and an uncomfortable heaviness in the chest. The latter is often accompanied by such vaso-motor symptoms as flushing of the face, cold hands and feet. At times her heart beats very fast.

**Examination.**—The teeth and tonsils are normal as you see. There is a slight soft enlargement of the thyroid gland. The systolic blood pressure is 140; the diastolic, 60. The cardiac rate is 112 per minute and regular. You note that when the patient holds her breath at the end of a deep inspiration, there is a decrease in the cardiac rate of from 10 to 20 beats per minute. You observe even from a distance, the cardiac hyperactivity in the apical region. Upon palpation you feel a thrill with the systolic impact, due to ventricular hyperactivity. Corresponding to this, upon auscultation the first heart sound seems somewhat thrill-like and split. The knee reflexes are very lively; the abdominal examination reveals nothing abnormal. The orthodiascopic tracing being passed around shows a heart of normal size and form.

The fluoroscopic examination showed over-action of the aorta, pulmonary artery and left ventricle.

As in Case 47, I do not believe that this patient is suffering from organic cardio-vascular disease, despite the abnormal physical signs upon auscultation and palpation.

In Case 47, the original cardiac insult was overexertion from horseback riding. After a quiescent period of several months, extrasystoles and cardiac pains reappeared following a Turkish bath. The sweating probably acted by exciting the vaso-motor mechanism which in its turn upset the as yet scarcely stabilized cardiac mechanism. The extrasystoles and cardiac pains were the main symptoms. In Case 48 there is a possibility that the history of severe hemicrania has some bearing as showing a general neurotic tendency. The history of dragging her child for several hours on a sled leaves no doubt that this overexertion was the immediate cause of the cardiac symptoms. The cardiac irritability has shown itself by tachycardia, dyspnœa and precordial heaviness, symptoms and signs similar to those found in the so-called soldier's heart.

Although the manifestations in Cases 47 and 48 are quite different, they both represent a type in which cardiac hyperirritability originates from cardiac overstrain. It is as yet a difficult and problematical matter to determine how or in what manner such overstrain produces cardiac neuroses. It is usually assumed that the onset of these attacks is marked by cardiac dilatation. Unless the usual accompanying symptoms of cardiac dilatation: Pulmonary edema, dyspnœa and cardiac failure are present, it seems quite difficult to support this hypothesis. It appears to me that perhaps physical cardiac overstrain may so affect the nerves in and about the heart, especially the nerve plexuses surrounding the aorta, that the cardio-inhibitory and the vaso-motor mechanism becomes reflexly and morbidly excited, with the resultant train of symptoms known as the irritable heart. In other words, unless actual cardiac dilatation can be proved, the primary result of cardiac overstrain may be a neurological, rather than a myocardial "insult." What influence, if any, slight mechanical stretching of the cardiac musculature may have in the production of symptoms such as outlined in these two cases is as yet unknown. It seems possible nevertheless, that such overstretching of cardiac fibers can take place without the production of the usual symptoms of acute cardiac dilatation with its decompensation.

**Prognosis.**—Case 47 had improved and was practically well, until the symptoms recurred after the Turkish bath. The chances are therefore that he will again improve rapidly, especially since the present symptoms are mild. Case 48 will probably run a longer and more protracted course because of the continuance and violence of her symptoms and because her housework does not allow her to take sufficient mental or physical relaxation and rest. The bromides are of value in both; and in both the extract of suprarenal gland in two grain doses is of benefit for the attacks of tachycardia and for the extra-

systoles. The drug is to be given at the time the symptoms are most violent. Digitalis is of no use in either case. Nor is rest in bed of any value except perhaps for a short time when the symptoms are most marked. In similar cases graded games in the form of play usually offer a happy combination of the proper amount of exercise with mental relaxation.

### CASES 49–54.—CARDIAC NEUROSIS (ESPECIALLY TACHYCARDIA) FOLLOWING FEBRILE CRISES—GENERAL COMMENT

Case 49.—Miss M. S., aged 38, had a mild influenzal attack three months ago. She had very slight fever but had frequent, long continued sweating even after her cold was better. Since that time, as she says, she "knows she has a heart." When trying to do her work as teacher, her heart "thumps," sometimes fast, sometimes slowly but always "accentuated," as she describes it. She woke up some weeks ago with a "thumping heart." She has recently suffered from slight indigestion with fulness in the stomach and eructations. She has never had rheumatism or scarlet fever and only occasional sore throats.

**Examination.**—The systolic blood pressure is 110; the diastolic, 80. As you see the thyroid is not enlarged. You notice that even now, although the heart rate is normal, the patient has slight dyspnœa. She is quite thin but she says that she has not lost any weight during the last year. As you listen to her heart, you find that the sounds are normal, with the exception of some exaggeration of the first sound at the apex. Cardiac inspection and palpation reveal nothing abnormal. As the patient walks about, there is no increase in the cardiac rate, although the patient states that she is now conscious of her heart action. Abdominal examination reveals nothing abnormal. There is no edema of the legs. The knee reflexes are normal, the urine is also normal. The orthodiascopic tracing, which I pass around, shows a somewhat narrow heart but not abnormally so for this thin-chested individual.

The diagnosis is plain. There is no cardio-vascular disease. The patient has a cardiac neurosis in which the prominent symptom is the annoying consciousness of her heart action. I believe the primary cause of the neurosis is the critical sweating which followed a mild influenza attack a few months ago. I shall discuss that factor in detail after I have shown you similar cases. I shall advise the patient to go back to her teaching and not be frightened by the peculiar sensations produced by her heart. She will be given the mixed bromides and the extract of suprarenal gland.

**Later Examination.**—You no doubt recall that I presented this patient two weeks ago. She now feels perfectly comfortable and is able to teach and walk as well as ever without any abnormal cardiac sensations. Indeed, as she puts it, she no longer knows nor feels that she has a heart. The medication consisted of 15 grains of the triple bromides given twice daily regularly

for one week, and five grain tablets of the extract of suprarenal gland, taken only when she felt particularly uncomfortable.

CASE 50.—Miss B. S., aged 24, has always been somewhat nervous and irritable. About six months ago, during the summer, she had a severe attack of tonsillitis accompanied by high temperature and followed by critical sweats. Since then she has suffered from tachycardia; according to her doctor her heart rate often reaches 140 per minute. She has also had gastric disturbances for weeks, the chief symptoms of which were frequent belching and a feeling of epigastric distension. At present she feels very weak. She has been kept in bed for *four months* by her family physician who thinks that she has an organic heart lesion. For a short period she improved but now she feels as badly as ever. The main cardiac complaint at present is palpitation due to rapid heart action. She also has headache and shooting pains in the limbs. She has lost about 20 pounds since her illness. Her highest temperature since she has been in bed has been 100°.

**Examination.**—You note that the patient is thin. There is no exophthalmos or goitre. The teeth and pharynx are normal. You see even from a distance that there is ventricular overaction in the mitral region. The cardiac area seems normal to percussion. The cardiac rate is very peculiar; at times it is between sixty and seventy per minute accompanied by a thumping action of the heart; then, suddenly and without extrasystoles, the rate reaches 110 to 120 per minute, especially when the patient sits up. You notice that even the rather rapid heart rate is not accompanied by dyspnœa. Upon palpation you feel a somewhat thrill-like systolic shock over the apex even when the heart beats slowly. Upon auscultation you hear a soft systolic murmur over the mitral area. The abdominal examination reveals slight epigastric sensitiveness to pressure; the abdomen is otherwise normal. There is no edema of the legs. The urine is normal.

The treatment will consist of the administration of fifteen grains of the mixed bromides given at bed-time; of the suprarenal gland extract in five grain doses three times daily before meals, and of an ant-acid powder after meals.

**Later Examination.**—We examined this patient about six weeks ago. Her systolic blood pressure is now 140; the diastolic, 90. You observe that she has become stouter; the patient states that she has now regained her old weight. She no longer feels nervous, her only complaint being occasional belching. Upon examination you find that the cardiac rate is around 100, and that upon taking a deep breath and holding it, the rate is reduced to 70 or 80 per minute. There is no thrill nor cardiac hyperactivity. The patient has improved very much and sleeps very much better than she did formerly. I shall prescribe $\frac{1}{150}$ of a grain of atropine sulphate, three times daily before meals, and magnesium usta powder in half-teaspoonful doses three times a day after meals.

Despite the history of tonsillitis, or even assuming diphtheria which nitated the cardiac symptoms, I do not think that the patient has or ever

had myocarditis. Myocarditis associated with diphtheria is a very serious disease, gives rise to decompensation in its early stages and is often fatal. In this case the cardiac symptoms began with critical drops of temperature and accompanying sweats, and continued for several months until rational treatment was begun. The benign course of the case, especially the absence of fever, would almost of itself make the diagnosis of actual cardiac disease highly improbable. It is interesting to note that the physician who treated the patient was apparently led astray in the diagnosis by the abnormal physical signs which were present; the thrill and tachycardia. As I have just pointed out to you, these have now disappeared.

General comment will be reserved until all the patients of this group have been examined.

CASE 51.—Mrs. T., aged 34, is married five years. Her husband had lues some years ago. His Wassermann blood reaction is now 2 plus. Four months ago while in the seventh month of pregnancy, she had a very severe attack of influenzal pneumonia. She was very ill for a long time; in fact, her life was despaired of. Convalesence was accompanied by severe night sweats. Even now, four months after the pneumonia, she still suffers from occasional sweats. Before her pneumonia she was always athletic and active. At present, attempts at walking or at lifting anything heavy cause severe pains under the left breast.

**Examination.**—You see that the patient is a very strong and vigorously built woman, in fact she looks the picture of health. She does not suffer from any dyspnœa while at rest. The thyroid gland is not enlarged. The systolic blood pressure is 140; the diastolic, 90. Upon precordial inspection and palpation, slight ventricular hyperactivity is demonstrable in the apical region. You also note that there is no sensitiveness upon precordial pressure. The abdominal examination reveals nothing abnormal. There is no edema of the legs; the urine is normal. I want you especially to note the orthodiascopic tracing which I pass around; it shows that the aorta is normal in size and form and that the general configuration of the heart is normal. This is important in view of the husband's history of syphilis and of the moderately strong positive Wassermann reaction of his blood.

Despite the history of syphilis in the husband, and the patient's cardiac complaints, I find no evidence of cardiac disease. Indeed, although her blood has not yet been examined for syphilis, even if it should prove mildly positive I would search for the cause of such reaction in some other organ. The history points definitely to the fact that the cardiac symptoms—palpitation and pain—came at the time of the critical sweats. I believe that this patient is another example of cardiac neurosis without actual disease.

**Later Examination.**—This robust patient you saw four months ago. She was given the extract of suprarenal gland, with bromides at night. Very soon after medication was begun the cardiac symptoms disappeared. She

was in the country, has since been active, and has felt well until about a week ago. She then developed very slight coryza with slight rise of temperature. Within a week, the old cardiac symptoms began, especially the precordial pains. These became so severe that at times she was unable to sleep. The cardio-vascular examination, as before, shows no abnormality of any kind. I believe that the slight cold acted anaphylactically, so to speak, and caused the present precordial attacks. In other words, she had slight sweats following the cold, and these acted as a sensitizer, as it were, in causing a relapse of the cardio-neurotic symptoms. I shall prescribe atropine sulphate in small doses. I believe that very soon she will again be quite comfortable.

CASE 52.—Mrs. S., aged 28, had influenza three months ago. This was followed by critical sweats for two weeks. She then developed rapid heart action and precordial pressure, especially when excited. Her appetite is fair. The chief complaints are throbbing temples and palpitation.

You see that at present the patient is slightly flushed. There is some throbbing of the carotids. There is no goiter or exophthalmos. The pulse rate varies rather rapidly from 90 to 110 per minute. There is a slight precordial thrill to be felt upon palpation, and a soft systolic murmur to be heard at the apex. The blood pressure is normal. There is no edema of the legs. When the patient holds her breath at the end of a deep inspiration there is a momentary but decided decrease of the cardiac rate. The knee reflexes are very lively. The abdominal examination reveals nothing abnormal. The urine is normal.

I do not believe that this patient has any cardiac disease. She represents another instance of continued tachycardia following the critical sweats incidental to most pneumonia attacks. Therapeutically I would suggest trying to build this patient up—she looks somewhat thin, you see—by giving her carbohydrates and fats. I shall prescribe the mixed bromides three times daily after meals, and the extract of suprarenal gland, to be taken when palpitation is particularly severe.

**Later Report.**—Her family physician reported to me that the patient became very comfortable after one week and that she has no tachycardia at present. She is able to go about, to walk and climb stairs, and is very comfortable in every way. Perhaps the assurance to the patient that she had no cardiac disease was a large factor in the rapid cure.

CASE 53.—Mrs. S., aged 28, had influenzal pneumonia three months ago. She was in bed for one month. Following the pneumonia, she had severe sweats for two weeks. Since then she has suffered from rapid heart action and palpitation, especially after exertion. She says that her appetite is poor.

**Examination.**—You notice that the patient is somewhat thin. She has no dyspnœa while at rest. The systolic and diastolic blood pressures are normal. Aside from a soft systolic murmur at the apex, the cardiac examination reveals nothing of note. When sitting quietly, the pulse rate is about

90 per minute; when walking about the room, it rises to 110. This rapidity is temporarily controlled by having the patient take a long breath and holding it. You have of course observed that during the entire examination the patient is readily perturbed, that she cries readily and is very excitable.

The patient is certainly neurotic in the ordinary acceptation of the word. I believe, however, that it was the critical sweating following her influenza which probably caused the palpitation and tachycardia. There is no evidence of any cardio-vascular disease. Assurance of that fact to the patient will no doubt play a very large role in alleviating her symptoms and in curing her.

CASE 54.—Miss P., aged 20, has suffered for two years from occasional right-sided pain and indigestion. She has had no abdominal symptoms during the preceding three months. Five weeks ago she suffered from slight epistaxis and influenzal bronchitis. Since then she has complained considerably of weakness, anorexia, occasional sweating and tachycardia. Her family physician states that her temperature has been normal and that at present she has no expectoration.

**Examination.**—You observe that the teeth and throat are normal. You hear a slight cardio-respiratory murmur over the pulmonary artery. The cardiac rate is 100 per minute; otherwise the cardiac examination reveals nothing abnormal. The lungs are clear. Upon abdominal examination you can feel a somewhat loose right kidney. This finding is by no means uncommon, especially in thin individuals. Despite the history of right sided pain, there is at present no tenderness over the appendix.

I believe that organic cardio-vascular disease can be excluded. Except for slight tachycardia, there are no abnormal cardiac signs or symptoms. The family physician has kept this patient in bed. I think she will do very much better if she gets up and goes about, resting in bed only for one hour after meals. Patients of this kind often feel exhausted and therefore need an abnormal amount of physical rest. She should be over-fed. A tonic pill containing iron, arsenic and strychnine is indicated. The extract of the suprarenal gland in five grain doses three times a day after her meals would probably help in controlling the tachycardia. The prognosis is excellent.

**General Comment.**—I have grouped these post-febrile tachycardia cases for you, gentlemen, because they are quite frequent and especially because tachycardia following any infection is commonly regarded as the result of organic cardio-vascular disease, particularly of the myocardium. One of the chief causes for this mistake has been that the etiological factor has not been properly traced. The usual teaching is that pneumonia or other febrile invasions attack the cardiac musculature and thus bring about actual myocarditis. That such organic changes can follow pneumonia is of course true. I believe that they are very rare, however, and that when they do occur the

patients do not exhibit symptoms similar to the cases I have just shown you. All of the cases that I have presented I consider functional in nature. By functional I mean that there are no organic changes in the cardiac valves, endocardium or musculature. Indeed I have dwelt upon the fact that the physical signs are often quite similar to, or identical with those found in cardiac neurosis from other causes, as for example the so-called soldier's heart. In the latter it is now generally conceded that organic disease is very rarely present.

What is the cause of cardiac irritability following critical defervesence? I shall not here speculate upon the mechanism of the crises themselves but I wish particularly to emphasize that the salient clinical feature is the sweating. As you know, taken by itself, this is an evidence of excitation of the sympathetic nervous system. If any of you have ever had a severe cold you will know that the sweats frequently continue for several days or even weeks after the cold has run its course. And just as the sweats are due to hyperexcitation of the sympathetic nervous system, so I conceive the cardiac irritability, especially the tachycardia, to be due to a concomitant hyperexcitation of the accelerator mechanism of the heart. I wish also to dwell upon the important clinical fact that once the cardiac neurosis has started, it may continue a long time after the sweating has ceased. Unless one conceives the primary cause in the light of an abnormal excitation of the sympathetics and accelerators, I believe it is exceedingly difficult to understand how tachycardia of this type can be brought about.

If one regards these cases as actual myocarditis with tachycardia, then, may I ask, how can such myocarditis suddenly cease in those in whom tachycardia suddenly stops? According to my assumption, an "insult" to the sympathetic nervous system may abnormally excite heart action for a longer or shorter time. It is in this manner that I view the etiology of the vast majority of post-febrile tachycardias. What there is in the individual that produces tachycardia in some and not in others I find it impossible to state, except the generic statement that different types of individuals react differently to what are apparently similar causes. We also know that the nerve balance of some individuals is very readily upset while others apparently show no such reaction. In addition, certain types of individuals may be called sympathetic-excitive in that their sympathetic nervous systems are especially irritable, while others may be termed vago-excitive because in them the vague nerves are hyperirritable.

I have often speculated upon the cause of the good results following the use of the extract of the suprarenal gland in the group of cases presented to you to-day. *A priori* one would expect that this drug, itself an excitant of the sympathetic nervous system, would be followed by poor results. I believe its great benefit depends upon the fact that it stabilizes the vasomotor system, thus tending to make it less hypersusceptible to abnormal nerve influences.

## CASE 55.—IRRITABLE HEART IN A PATIENT WITH PREVIOUS PAROXYSMAL TACHYCARDIA

O. K., male, unmarried, aged 57, in previous years had always been active and athletic. About twenty years ago he felt something "snap" in his heart, as he described it. This "snap" was followed by very rapid heart action which as suddenly stopped. It was not accompanied by any gastric symptoms. He does not know the cause of the original attack. Following the first attack, he had many others lasting from one half hour to one day. The attacks occurred at varying intervals of from one month to one year. Several months ago, he developed gastric symptoms for the first time; namely, poor appetite and distention after meals. He feels very "nervous" and says he cannot sleep at night. Recently his heart action began to annoy him; he has a feeling of palpitation and sometimes his heart "stops." After stopping it occasionally pumps hard.

**Examination.**—Let me first give you the result of the gastric analysis: There is hypersecretion but no hyperacidity: There is no evidence of gastric ulcer. You see that the patient though small, is wiry and is very well preserved for his age. The systolic and diastolic blood pressures are normal. Auscultation reveals no abnormal heart sounds. Palpation over the heart is also normal. The orthodiascopic tracing which is being passed around shows a heart normal in size and form. Of most interest is the cardiac rate. At times it is rather rapid, about 100 a minute, then it suddenly changes to a normal rate. In addition the rhythm is occasionally disturbed by extrasystoles. The patient's tongue is somewhat coated. There is no edema of the legs. The urine contains a goodly trace of indican, it is otherwise normal. It is of interest to speculate upon the influence of the prior paroxysmal tachycardia on the origin of the extrasystoles which are now present. A gastric neurosis such as this patient has is in itself sufficient to produce reflex excitation of the heart nerves, with resultant tachycardia and extrasystoles. But in view of the old paroxysmal tachycardia, one may assume that the patient or rather his cardiac nerves have become hypersusceptible, so that now they are more readily influenced and upset by any abnormal reflex excitation from a neighboring organ, in this instance, the stomach.

What this patient especially requires is mental relaxation. He has been worried about his work, he feels that because of his heart he may not be able to continue at his business as a boat-builder, and this doubtless has an influence in causing the sleeplessness and gastric symptoms. I would advise him to seek some relaxation such as fishing, and to give up his work entirely for a week or two. He should be given bromides for his sleeplessness and atropine sulphate in small doses three times a day in order to try to control the cardiac irregularity.

**Later Report.**—I have heard from the patient's physician that he is much improved, and that he sleeps better. The extrasystoles are still present,

but they no longer annoy him, and he is again partly able to take up his old work as boat-builder.

### CASES 56, 57.—CARDIAC NEUROSIS FOLLOWING GASTRO-INTESTINAL DISTURBANCE

CASE 56.—M. W., female, aged 41, had always been in the habit of bolting her food: She suffered frequently from belching. The patient states that acids and sour things do not agree with her. She has two children, her menses are regular. Some weeks ago, after bolting a heavy meal she had what she called a "gas attack:" Her abdomen felt tense and she belched considerably. The attack was accompanied by palpitation. Her family physician who saw her at the time stated that he found the heart action very irregular. After one week he noted that the heart became more regular. There is no history of rheumatic or other infection.

**Examination.**—Upon examining the mouth of this patient, you find that the teeth and throat are normal. The systolic blood pressure is 160; the diastolic, 100. There is, as you see, no thyroid enlargement. The heart is rapid and regular, its rate is 100 per minute; this rapidity may be due to her evident nervousness at present. There is a somewhat thrill-like apical first sound to be heard, and a thrill-like systolic shock to be felt at the apex; they are probably due to rapid and somewhat over-active heart action, for if the cardiac rate be decreased by having the patient take a deep breath and holding it, you find that both these cardiac abnormal features disappear. The orthodiascopic tracing which I am passing around shows a heart normal in size and form. The abdominal examination reveals nothing abnormal. The knee reflexes are normal. There is no edema of the legs. The urine is normal.

I believe we can exclude organic cardiac disease despite the abnormal thrills heard and felt over the apex. I have shown you many similar cases to prove that these abnormalities are very often of functional origin and due to an over-rapid or an over-active heart. Although there is no cardiac irregularity at present, the family physician's description of the arrhythmia at the time he examined her leaves no doubt in my mind that it was due to extrasystoles. These have now apparently entirely disappeared but the patient still complains of a subjective feeling of "palpitation." I believe that both the extrasystoles and "palpitation" are caused by reflex disturbance of the cardiac mechanism from the attack of indigestion. Such cardiac neuroses are sometimes regarded as toxic in origin. I assume this implies that a certain amount of poisonous material is absorbed into the system and that this deleteriously affects the cardiac inhibitory apparatus or the heart muscle itself. In my opinion, the vast majority of these disturbances of cardiac rhythm are not toxic in nature but are simply due to reflex excitation of the cardiac nerves following any stomach disturbance. I have found extrasystoles frequently in patients who are subject to belching and gastric distension. They are less

frequent, I believe in those with actual organic gastro-intestinal disease than in those with functional derangements.

I shall prescribe atropine sulphate three times daily in doses of 1/100 of a grain, and an ant-acid powder to be taken after meals; she should also be given 30 grains of the triple bromides before retiring at night.

**Later Report.**—I have since heard from the family physician that the patient is feeling very well, and that the subjective feeling of palpitation and the arrhythmia have entirely disappeared.

CASE 57.—S. B., female, aged 27, gives the following history. Several months ago she was shocked by the unexpected death of a friend. A few days thereafter she had nausea and vomiting for which she remained in bed two days. She then felt well for six weeks. Her family physician stated that she later had a hysterical vomiting spell and that the vomiting lasted five days. She remained well until three weeks ago. Then after eating some "fried stuff," as she calls it, she became nauseated, and heard noises in her ears for several days. Very soon thereafter she complained of tachycardia, of extreme dizziness and of frequent belching. Her bowels are constipated; her appetite is fair. At the present time her chief complaints are almost constant tachycardia and dizziness.

**Examination.**—The patient as you notice, gives the impression of being very nervous; she is restive and her face is flushed. There is no enlargement of the thyroid gland. The systolic blood pressure is 110, the diastolic, 70. The cardiac rate is 110 per minute. When she holds her breath at the end of a long inspiration, you find that the rate occasionally becomes normal in rapidity. Her hands are cold. Except for the abnormal rapidity, the heart is normal upon auscultation and palpation. The knee reflexes are very lively. There is no edema of the legs. The orthodiascopic outline of the heart, as you see from this tracing, shows no abnormality of any kind. Upon neurological examination, I find there is no ataxia of the upper extremities, nor any differences in cutaneous sensibility to touch, heat or cold. The ophthalmological examination reveals concentric contraction of both visual fields.

I do not find any evidence of organic cardio-vascular disease. This patient is a very high-strung woman, and it appears that her entire nervous system became upset from the nervous shock of the death of her friend. Thereafter she had several gastric attacks, and it seems as if the last spell of indigestion disturbed the nervous control of the heart and produced the tachycardia of which she has since complained. It is very important to determine the cause of the dizziness, especially in view of the ocular findings, for some cerebral growth may possibly cause both the dizziness and the contracted visual fields. From the cardio-vascular symptoms, however, I am much more inclined to the view that vaso-motor changes in the cerebrum are responsible for the dizziness, the frequent noises of which she complains, and even for the contracted visual fields. At any rate, for the present, I shall proceed upon this hypothesis and shall treat her accordingly. I shall pre-

scribe atropine sulphate three times a day to be taken before meals, bicarbonate of soda after meals, large doses of the mixed bromides each morning, and the extract of suprarenal gland in one grain doses for severe attacks of dizziness and tachycardia.

**Later Examination.**—You recall we saw this patient about two months ago. At first she got along very slowly, dizziness and palpitation still being the main symptoms. She gradually regained confidence in herself, and with this return of confidence, dizziness and tachycardia gradually diminished. She finally went to the seashore, where after a time she took ocean baths. You notice the marked difference, both in her appearance and in her general nervous condition. She has gained very much in weight. If you examine her heart you will find that the rate is almost normal. She no longer complains of dizziness and not only feels better, but as she puts it, she is perfectly well.

This case may be coupled with the preceding one as another instance of cardiac neurosis following gastric disturbance in a particularly neurotic individual. Both cases show how readily the normal cardiac mechanism may be upset by reflex disturbances from a disordered stomach, particularly in hypersusceptible, neurotic patients.

### CASE 58.—CARDIAC NEUROSIS—SUBJECTIVE DYSPNŒA

M. L., female, aged 39, has had five children and no miscarriages. Her menses are regular. She herself states that she is of an "hysterical temperament." She says that about twelve years ago she heard of the sudden death of a child. Since then, when aggravated by any quarrel, she feels "palpitation and tightness in the heart." About two years ago, after some trouble with her grown-up son, she was considerably upset; this was followed by pain and abdominal colic. She does her own housework, is very exact about it, but feels exhausted after doing it. She weighed 200 pounds some years ago, she now weighs 185. Besides the abnormal sensations in the heart, she complains of a choking feeling in her throat.

**Examination.**—You observe that this patient has no dyspnœa while at rest. Her teeth and tonsils are normal. The systolic blood pressure is 130; the diastolic, 90. The heart, you find, is normal to auscultation and palpation. The cardiac rate is absolutely normal, indeed, there is no increase of the cardiac rate even when the patient walks around the room rapidly. The orthodiascopic tracing, you observe, shows a rather small but flat-lying heart. Its posture on the diaphragm is no doubt due to the fact that the diaphragm has pushed up the left ventricle. The abdominal examination reveals nothing abnormal. The urine and knee reflexes are normal.

I demonstrate this case to you, not because the patient has any cardiac disease, but because she is one of a large group found frequently in private practice, who complain of all sorts of subjective sensations in the chest—feel-

ings of unrest, palpitation without actual tachycardia, a choking and gripping sensation etc.— yet who have no actual cardiac disease. A neurotic insult, so to speak, plays a large role in producing an outbreak of such symptoms. As you see, some special cause can usually be elicited by a careful history. Reassurance and suggestion play a very large role in the therapy of these cases. They require mental relaxation rather than physical rest. If treatment along such lines be carried out, almost miraculous and rapid cures can be accomplished.

### CASE 59.—CARDIAC NEUROSIS AND PREGNANCY

Mrs. M. R., aged 35, tells us that she has been nervous for years. She is now in the third month of her fifth pregnancy. In previous years when upset from any cause, she suffered from tachycardia. Of late months she has had occasional pains in the left chest, anteriorly and posteriorly. There had also been a recent recurrence of old gastric symptoms, chiefly belching; these have ceased since her pregnancy. She has had "palpitation" off and on for several weeks; she is very much worried now because she has been told that she has "heart disease."

**Examination.**—The teeth, throat and thyroid as you see, are normal. The systolic blood pressure is 143; the diastolic, 88. The cardiac rate is 100 per minute. You observe that holding the breath at the end of inspiration has a marked effect in slowing the heart rate. Upon palpation over the heart you feel a slight systolic thrill at the apex; a soft systolic murmur is audible over the same area. Abdominal examination reveals nothing abnormal. The urine is normal. There is no edema of the legs. The knee reflexes are very lively. The orthodiascopic tracing being passed around shows a normal-sized heart and aorta. Upon carefully palpating the left chest, you will find two small sensitive areas in the fifth intercostal space, one anteriorly, the other posteriorly, which make the patient wince as you press upon them: These are the usual sensitive spots found in intercostal neuralgia.

I find no evidence of any cardio-vascular disease. The patient has a mild intercostal neuralgia. The localized pains, the moderate tachycardia, and probably also the soft apical murmur and thrill have apparently been interpreted by another physician as "heart disease." The latter diagnosis was told the patient. She naturally became frightened, and being of a neurotic temperament it intensified her nervousness. There is no indication for the interruption of pregnancy. The chief "remedy" is assurance to the patient that she has no "heart disease." There is no reason to fear that the mild tachycardia will interfere with the normal course of pregnancy or parturition.

### CASE 60.—RECURRENCE OF SYMPTOMS OF EXOPHTHALMIC GOITER AT THE MENOPAUSE

Mrs. O., aged 49, has had two children; the younger is 25 years old. About 18 years ago she had exophthalmic goiter with typical symptoms:

Tremor, nervousness, enlarged thyroid and "skipping of the heart." A thyroidectomy was done with excellent results. She felt well for many years. About three years ago she complained of belching, intestinal rumbling, pain across the chest, faintness and dizziness. These symptoms have usually been temporarily relieved by gastric eructations. Until two months ago her menses were regular. Latterly, after such slight exertion as walking, she has suffered from midsternal pains radiating to both arms; she feels weak and has "skipping" of the heart. In addition she occasionally has a burning sensation in the throat. All these symptoms are usually relieved by belching. There is no vomiting or regurgitation of fluid. The bowels are regular. She has been eating rather heavily of late. The urine contains a trace of sugar; it is otherwise normal.

**Examination.**—You note the scar in the neck due to the thyroidectomy. There is no exophthalmos. There is some carotid overaction. You feel a strong, thrill-like impulse at the apex. Upon auscultation over the mitral area, there is a broken, cog-like systolic first sound. The other cardiac sounds are normal. The cardiac rate is 100 per minute, and is interrupted by occasional extrasystoles. The systolic blood pressure is 160; the diastolic, 70. The abdomen is not sensitive to pressure. The margin of the liver cannot be felt. The orthodiascopic tracing, as you see, shows an enlarged aorta as well as enlargement of the left ventricle below and to the left. There is no edema of the legs. The knee reflexes are lively.

I shall first discuss the physical signs at the apex: The palpable thrill and the broken, thrill-like first sound. These are by no means indicative of a valvular lesion. Such abnormal sounds are common in rapidly beating, violently acting hearts found in typical exophthalmic goiter. They are also common in the irritable heart observed in general and military practice.

Now, with reference to the orthodiascopic findings. Is the enlarged left ventricle the result of dilation or of hypertrophy? The form of the heart in the tracing is not characteristic of hypertrophy; it has not the large, obtuse, egg-shaped form. When I looked at the heart through the fluoroscope, I missed the rocking action like a see-saw with the fulcrum in the region of the pulmonary artery. This motion is characteristic of the hypertrophied left ventricle. Ventricular enlargement such as you see in this tracing is not uncommon in exophthalmic goiter. In the earlier stages of the disease, it can be scarcely attributed to hypertrophy, for post-mortem examination has shown that hypertrophy of the cardiac musculature is rare. I believe the aortic enlargement visible in the tracing is due to dilatation of the artery, probably from the violence with which the blood is thrown into the circulation.

How shall we interpret and correlate the midsternal pains, the belching and the exhaustion? The "skipping" of the heart complained of by the patient is readily explained as due to the extrasystoles which we discovered upon examination. I believe the pain and the gastric symptoms, especially

the relief following belching, are due to cardiospasm. The cardiac irregularity may have its reflex origin from that condition.

I consider that the cardiac and gastric symptoms are the result of a neurosis accompanying the beginning menopause. In fact, the present symptom complex may be considered, in a sense, a recrudescence of the old goiter symptoms; but this time they are of climacteric origin. The therapy should be directed mainly to the gastro-intestinal canal. The patient should be given an ant-acid powder. She should take simple food frequently rather than regular three large meals, and should be given bromides at night.

**Later Report.**—Three weeks later her physician stated that she was much improved and that she could walk without pains or discomfort.

### CASE 61.—EXOPHTHALMIC GOITER WITH HYPERTENSION

P. G., aged 27, has been married one year. The menses had been regular until six weeks ago. She says she had a goiter and occasional tachycardia for some time before marriage. For the first few months after marriage she felt well. Latterly she has been tired and "nervous" and has lost weight.

**Examination.**—I present the patient to you as a typical example of exophthalmic goiter with a so-called goitrous heart. You observe the moderate exophthalmos, the flushed facies, the tremor of the outstretched hands, the enlarged thyroid. You can also see the tachycardia by watching the rapidly throbbing carotids, as well as the heave of the left breast with each ventricular systole. The heart rate is 120 per minute. You feel a decided systolic thrill at the apex, and you hear a broken, split, thrill-like first sound over the mitral area. These abnormal cardiac signs are not necessarily evidence of valvular or myocardial disease; they are probably due to the violence with which the blood is thrown into the aorta during systole.

The orthodiascopic tracing, you observe, shows an enlarged aorta; the ventricular outline is normal in size and form.

I present this case to you chiefly because of the marked hypertension. The systolic blood pressure, you observe, is 210; the diastolic, 50. The urine is normal in every respect. There is no edema of the legs Moderate hypertension is part of the clinical complex of exophthalmic goiter. Occasionally, however, as in this instance hypertension is extreme. But this is not indicative of renal involvement in patients with exophthalmic goiter; it is probably due to an extremely hypersensitive and labile vaso-motor system which reacts in an exaggerated fashion to any abnormal stimulus.

It is a matter of interest to speculate as to whether marriage had any relation to the onset of the goitrous symptoms in our patient. From the history it is probable that a mild non-toxic goiter was present before marriage. We know that pregnancy does occasionally activate a dormant goiter, apparently by its as yet somewhat mysterious hormonic endocrine influence upon the thyroid. However, since it is only six weeks from the date of her last

menstruation, and since goitrous symptoms have been present several months, pregnancy can be excluded etiologically. Is it possible that marital intercourse of itself can so affect ovarian function as to profoundly and deleteriously influence the entire endocrine system, and with it the thyroid, thus causing the complex of exophthalmic goiter? Although we are now delving into the unknown, it seems possible that sexual intercourse can effect an already pathological thyroid in the manner just suggested. On the other hand, it must be remembered that sexual intercourse and excitement as such, may also conceivably exert a morbid influence upon patients with goitrous tendencies by producing general neurotic hyperexcitability.

**Therapy.**—Should the patient be pregnant, I would insist upon immediate emptying of the uterus. This can and should be done, I believe, under morphine and not general narcosis. The remainder of the treatment should consist essentially in rest, attempts at overfeeding and in the administration of large doses of mixed bromides. Intercourse should be interdicted for several months at least. Cases such as these run a very protracted course; they are very trying both to physician and patient, for care and supervision must be exercised for months before marked or indeed any improvement becomes evident. Even when these individuals are apparently cured, they are apt to have mild or severe relapses if their lives are not carefully regulated, especially in the avoidance of all kinds of excitement. I shall not take up the advisability of thyroidectomy in this case because it would lead us too far afield.

### CASES 62–64.—TOBACCO HEART—GENERAL COMMENT

Gentlemen, I have grouped for you three cases of so-called tobacco heart whose histories and examinations we shall first complete; we shall then discuss the etiological and various other factors.

Case 62.—E. C., male, aged 59, a baker, has always been a heavy cigarette smoker, often smoking from forty to fifty cigarettes daily. He says that he is of a very excitable temperament and easily aggravated. About a year and a half ago, following a dispute, he had his first attack of precordial pain. Since then these pains have been very frequent, especially when walking or driving in the open air. In his bakery he can walk around very rapidly without discomfort of any kind. Occasionaly the precordial pains radiate to the left shoulder. He has not lost weight, he has no gastric or other symptoms.

**Examination.**—You note that when the patient is sitting quietly he is not dyspnœic nor does he become dyspnœic when walking about the room. The systolic blood pressure is 120; the diastolic, 80. You observe that neither upon palpation, percussion nor auscultation does the cardiac examination reveal any abnormality, whether the patient be at rest, walking, sitting quietly or lying down. The knee reflexes are very lively. The abdominal examina-

tion reveals nothing abnormal. There is no edema of the legs. The urine is normal. Despite the fact that this man is broad-chested, the size of the aorta and of the heart as shown by the orthodiascopic tracing being passed around is surprisingly small.

In my opinion the patient presents no evidence of organic cardio-vascular disease. I believe that the cardio-vascular symptoms can be explained as the result of tabagism in a person of neurotic tendencies.

**Later Examination.**—This patient you saw about four weeks ago. I presented him as a case of "smoker's heart." At this examination you will discover that the blood pressure and other cardiac findings are the same as at the first examination. The patient feels very much better, is able to walk in the open, and says he can walk even in cold weather without precordial pains. I had given him the mixed bromides in 15 grain doses mornings and evenings, and a tablet of nitro-glycerine and atropine sulphate three times daily before meals. For attacks of precordial pain he took the extract of suprarenal gland.

Case 63.—A. B., aged 54, a physician, gives the following history. Except for articular rheumatism some twenty years ago and for occasional attacks of lumbago, he has had no antecedent venereal or other infection. He was a light smoker until ten years ago; he then began smoking heavily, consuming as much as one half dozen cigars and ten to fifteen cigarettes daily. He has had occasional slight gastric hyperacidity and vertigo for eight years; both were usually relieved temporarily by belching. About five years ago he had a sudden severe attack of nausea and vomiting, followed by severe vertigo which lasted four weeks. The attack was regarded as due to over-work and nervousness. He stopped eating seasoned food. Since several months he has had severe midsternal burning pains radiating to the left forearm and fingers. When the pains occur while walking, they are sufficiently intense to make him stop. The attacks are occasionally accompanied by nausea. They usually last two or three minutes. They are not accompanied by dyspnœa or palpitation. The pains are most apt to occur when he starts walking after a heavy meal. With the cessation of pain, the patient can again walk comfortably and even play golf. He has not lost weight, his appetite is good, all other functions seem to be normal.

**Examination.**—You note, gentlemen, that this large and robust-looking man rests very comfortably, showing no dyspnœa when sitting quietly or when walking about the room. The systolic blood-pressure is 140; the diastolic, 90. You find that a most careful examination of the heart by auscultation and palpation in the resting or lying position or after exercise, elicits no untoward sign or symptom. The urine is normal. The electrocardiogram being passed around shows nothing abnormal. The orthodiascopic tracing shows a normal sized aorta, the heart is somewhat broad, but not too much so for a man of his physique. The Wasserman blood test is negative. There is no edema of the legs.

Despite the presence of the precordial attacks from which this patient suffers, I feel quite sure that he has no cardio-vascular disease. I attribute even the gastric symptoms to the same cause that produced the precordial pains, namely tobacco poisoning.

**Later Examination.**—You recall this patient and his history, and that he was here one week ago. You will find upon examination that there is no change in the cardiac status. The blood pressure is normal and all the other physical signs are the same as previously. The patient feels very much better; he has cut down the amount of his smoking considerably. He finds that he can now walk comfortably, and that the midsternal and other pains are very much less. The only medication he is taking is atropine sulphate tablets in doses of $\frac{1}{150}$ of a grain three times daily before meals.

CASE 64.—M. K., aged 46, a butcher, was sent to us by a well-known gastro-enterologist. According to his report, the gastric findings after the extraction of a test meal show typical hyperacidity; there is no hyper-secretion, and no blood or mucus in the extracted meal. There is no blood in the stools after a meat free diet.

Regarding the history, the patient states that he has smoked since he was nine years old. When still a young man he consumed from fifteen to twenty cigarettes a day. There is no previous history of rheumatism or of any infection. He was always a quick and hearty eater, and a hard worker. About nine months ago, after pushing a very heavy meat cart, he was suddenly attacked with severe precordial pains. Since then any exercise, especially walking even slowly in the open air, produces midsternal pains which radiate to the back and arms. There is in addition slight midsternal sensitiveness during or immediately after an attack. Besides these chest symptoms, he suffers frequently from belching and faintness. He had been on an "ulcer diet" for several weeks. Once he broke his diet by eating cheese-cake and some sour food; this immediately produced very severe abdominal symptoms—colic, and a tympanitic and somewhat rigid abdomen, especially upon the right side. He often has sudden hunger pangs and complains of frequent and continued intestinal rumblings, probably due to hyper-peristalsis. The Wassermann blood reaction is negative.

**Examination.**—Inspection of the mouth, tonsils and gums reveals nothing abnormal. With the exception of a small area of midsternal sensitiveness, you will find that the cardiac examination reveals nothing abnormal upon the most careful inspection, palpation or auscultation. When the patient sits quietly, he complains of no pain, but as I allow him to walk around, even slowly, he begins to complain of his midsternal pain which as he says is beginning to shoot toward the back and the left arm. The present attack, the patient says, is somewhat exceptional in that it occurs indoors instead of in the open. When you compare the right and left radials, you find that the latter seems somewhat smaller than the right, yet despite this difference, the systolic blood-pressure of both brachials is equal; it is 145 in both. The

diastolic blood-pressure of the left arm is 84. The orthodiascopic tracing being passed around shows that the aorta is normal in size and form, that the left ventricle is small, and that the remainder of the cardiac outline is also normal in size and form. There is no edema of the legs. The urine is normal. The electrocardiogram, which I show you, is likewise normal.

Despite severe precordial pains, we find no tangible evidence of organic disease. I believe the patient is suffering from a cardiac neurosis due to tabagism. I want to emphasize the combination of gastric and cardiac symptoms in this case as in the other.

**Later Report.**—I received a report of this case from the family physician some months after we had examined him. The doctor stated that for some weeks, the pains remained very severe, despite the tablets of atropine sulphate and nitroglycerine which he took. Gradually, however, the pains became less acute and the patient was able to walk in the street in warm weather. Latterly, he still further improved in that he was able to drive his own automobile. My impression is that improvement will continue so that the patient will eventually be able to get about as comfortably as he did prior to his precordial attacks. However, he may never be able to again take up his laborious work.

**General Comment.**—There are two opposite views regarding the effect of tobacco poisoning upon the heart. In the one, the more popular view, nicotine, the main deleterious alkaloid of tobacco, is assumed to directly injure the cardiac structure, especially the myocardium, thus producing myocarditis. The other and less commonly accepted theory, the one to which I adhere, is that nicotine is a specific neurotropic poison which is especially harmful to the nerves of the heart. This theory is partly based upon the well known experimental physiological fact that nicotine injected into animals acts upon ganglia and nerve structures.

I have reviewed the existing experimental procedures that sought to produce an actual cardiac lesion in animals either by nicotine injections or by having animals inhale tobacco smoke for longer or shorter intervals. To say the least, I have found this evidence conflicting and of problematical value. The meagre pathological data relating to the human subject states that the heart of smokers who have died from other causes showed only slight muscle changes and that this was attributed merely to the incidental tachyardia.

A clinical argument against the presence of any severe organic cardiovascular change from nicotine is the fact that after the etiology is recognized and patients stop smoking, the symptoms usually recede, the precordial pains become less and the patients soon become more comfortable. It is interesting to note that most of the patients I have seen with so-called tobacco angina suffer from their precordial pains and disturbances chiefly when walking in the open, and not when walking about in a warm room. It seems probable that cold air causes some vaso-motor change which reflexly

excites the cardiac nerve control, produces coronary spasm and consequent precordial pains.

You will note in the cases that I have presented to you that there seems to be some culminating factor which starts cardiac pains and other symptoms. In the one case, it was pushing a heavy meat cart; in another, the doctor, it followed a severe nerve strain. In other words, anything which can throw a physical or nerve strain upon the circulation is apt to be the final factor which elicits cardiac symptoms. I have already stated that when smoking is stopped the patients are usually relieved of their cardiac and other symptoms. But this is by no means always the case; for as in other instances of a labile and disordered nervous mechanism, an injury or insult, once instituted, is sometimes apt to cause long continued symptoms for weeks or even months. This is well demonstrated in Case 64. This patient will probably go through many trials before his vaso-motor system is so stabilized that he can walk comfortably and without pain. Such patients must walk slowly at first and exercise only in their rooms; if there be no recurrence of severe pains, they may venture out in the open, but at the outset only in warm weather. It may take months until they, or rather, until their vaso-motor systems are so re-trained, that they can walk at their natural gaits in the open air. It takes even longer before they can take up more strenuous physical exertion, especially hard work. Here also re-training must proceed very slowly. Patients with sedentary occupations and severe tobacco symptoms should at first work but a few hours each day. Patients with laborious occupations will at the beginning have to seek some work which does not require severe physical exertion.

What is the fundamental cause of the precordial pains, the chief symptoms from which patients with "tobacco hearts" suffer? While my own theory of its causation is not susceptible to proof, it appears to me that the pains are produced only by marked interference with the coronary flow, similar to the mechanism assumed in the severe precordial so-called anginal attacks accompanying coronary embolism and thrombosis. I have emphasized and given my reasons for the statement that I do not regard actual cardiac or coronary disease present in smoker's hearts. Hence, according to my assumption, interference with the coronary circulation can only come from spasm of these vessels, a fact apparently depending upon abnormal excitation of the nerves controlling their caliber. Remember that nicotine is a neurotoxic and neurotropic alkaloid. We do not know the paths of the abnormal excitations which influence the intra-cardiac blood supply but for the present I believe that the clinical facts can be explained in no other way than by assuming interference with the coronary flow.

Therapeutically I have found doses of atropine sulphate given to the limit of tolerance the best single drug. This may be taken regularly for weeks or even for months; atropine poisoning can be avoided by searching for its well-known symptoms from time to time and then if necessary remitting the drug

for a few days. Different patients tolerate the drug differently, so it is always wise to start with small doses and increase it as rapidly as possible to its full physiological effect. When, in addition to the precordial pains, the patients are restive and irritable, occasional doses of the triple bromides have a good effect. For special attacks of pain, I have found the extract of the suprarenal gland of great benefit.

I want to recur for a moment to the gastric and enteric symptoms in two of our cases. I believe that in both excessive smoking was their cause. Just as nicotine can cause poisoning of the heart nerves, so I believe that it can occasionally cause poisoning of the gastric nerve supply, with attendant violent gastro-intestinal disturbances. Indeed, in Case 64, at one time the abdominal symptoms—very severe gastric and intestinal spasms—predominated the clinical picture. It seems probable that tabagism can produce hypeperistalsis, hyperacidity, gastrospasm, severe abdominal pains and other symptoms resembling gastric ulcer by its neurotropic effect upon the gastric and intestinal nerve supply.

The dizziness that many of the patients with tobacco hearts complain of is very probably connected with disturbance of the vaso-motor mechanism, thus affecting the blood supply to the cerebral center or centers which control equilibrium.

To sum up, while I cannot deny that tobacco poisoning may possibly, or to put it another way, can conceivably produce some organic change in the cardiac structures, I think that up to the present time, definite proof of such organic change is lacking, despite the many clinical and experimental papers published upon the subject. On the other hand, upon physiological and clinical grounds, I hold that all the symptoms can be accounted for on the assumption that the main tobacco alkaloid, nicotine, can act as an almost specific neurotropic poison, can thus affect the controlling nerve mechanism of the heart, and can consequently produce not only various types of cardiac irregularities but also very severe precordial distress from coronary spasm.

### CASES 65–67.—"IRRITABLE HEARTS" IN PATIENTS WITH ORGANIC VALVULAR DISEASE—GENERAL COMMENT

Case 65.—J. I., male, aged 15, has had frequent attacks of tonsillitis. As a young child he was a somnambulist. He has grown considerably during the past year. He was fairly athletic and always able to run at least a half mile without getting out of breath. Two weeks ago he developed fever which lasted one week and was accompanied by rapid heart action. A cardiac murmur was then detected for the first time. Since then any excitement or exertion, even walking, causes tachycardia.

**Examination.**—The boy, you observe, is quite tall for his age. The thyroid gland is not palpable. The teeth are in good condition. The tonsils are somewhat fleshy. Inspection of the precordium shows well marked

hyperactivity. There is no thrill or tenderness on palpation. Upon auscultation you hear a very faint systolic murmur when the boy sits up; this becomes quite loud when he is lying down. When the boy rests quietly, the cardiac rate is 90 per minute; after walking about the room once, the rate rises to 110 per minute, but the patient shows no sign of dyspnœa nor does he complain of the rapid heart action.

Now let us fluoroscope him. You observe first a heart normal in size and form; second, the ventricular hyperactivity. I wish you to observe carefully the effect of deep breathing upon the ventricular activity. He is now holding his breath at the end of a deep inspiration. You see not only a much slower cardiac rate, but also the deeper, quieter, less violent ventricular systoles. This again literally shows you what you have so often heard me describe as the effect of breathing on some rapidly acting hearts, namely that it not only slows the heart, but may also produce less violent ventricular contractions. The latter can sometimes be ascertained by the usual clinical methods of examination, but it is important to have an ocular demonstration by the X-ray in order to establish the validity of this phenomenon.

**Diagnosis.**—The first question to decide is whether the systolic murmur is the result of a mitral lesion or is functional in origin. As you know and as I have already demonstrated to you, loud systolic murmurs especially over the mitral area, are not infrequent in rapid heart action from any cause. I believe that in the present case the only decisive criteria indicating endocarditis are the frequent tonsillar attacks and the fact that the cardiac symptoms followed immediately after a febrile onset. We do not know the exact nature of the fever, it is true, but that it was probably not due to tonsillitis. When fever is part of the syndrome of a general bacteremia from any cause, the endocardium can become infected, often as a terminal event. There is of course no such evidence in our patient. Unless fever be of rheumatic, tonsillar or scarlatinal origin, I believe that it only rarely causes endocarditis. Hence, as a corollary, the vast majority of so-called febrile murmurs are not due to actual heart disease. Rheumatic fever occasionally produces only very mild, fleeting rheumatic manifestations, and almost immediately attacks the endocardium. I believe this boy represents this exceptional course, because the physical signs and manifestations of heart disease followed so directly upon the fever. I present him to you as an example of a mild, acute, mitral regurgitant lesion in which the main symptom is the tachycardia.

I believe that the prognosis is good because the boy looks well, has no fever now nor any subjective symptoms. I assume therefore that the pathological process is apt to stop soon, and to gradually retrogress so that in the course of years, or perhaps even of months, the physical signs of the lesion may entirely disappear. I believe this happens more frequently than is usually suspected. For example, I have examined the patients of careful dependable physicians who assured me that their patients had had typical rheumatic endocardial lesions in previous years, with classical physical signs;

yet upon more recent examination, the latter had entirely disappeared. Occasional autopsy examinations showing retrogressive changes in the mitral leaflets of those with typical rheumatic histories also support the view that prior active processes may ultimately retrogress to such an extent as to leave very little permanent evidence of the disease.

Another reason for presenting this patient is to show you how a heart with an organic lesion may become hyperirritable in the same manner as a heart with purely functional derangements.

**Later Report.**—I have heard from a sister of the boy four weeks after we had examined him that he was at the seashore, feeling well but that his heart action was still rapid. As I have stated to you, it may be months before the cardiac irritability quiets down and the heart rate again becomes normal. I prescribed the salicylate of soda and mixed bromides; these were to be taken intermittently for several months.

Case 66.—L. S., female, aged 11. Her physician states that she had a typical attack of cardiac decompensation two years ago: Edema of the lungs, enlarged liver and dyspnœa. This occurred soon after an attack of rheumatism. When a baby she had been subject to tonsillitis; a tonsillectomy had been performed a long time before cardiac symptoms began. Her cardiac complaints at present consist in the consciousness of rapid heart action, and in shortness of breath when climbing stairs, running or after skipping the rope. She is also subject to sudden severe attacks of hemicrania.

**Examination.**—The child you see is well developed. She is not dyspnœic when at rest. Inspection of the neck and chest reveal nothing abnormal. Upon palpation, you feel a systolic thrill-like precordial shock at the apex. Upon auscultation, you hear over the mitral area a loud systolic murmur which is transmitted to the left axilla, and posteriorly to the left scapular angle. You also hear a loud systolic murmur over the pulmonary artery; this is probably transmitted from the mitral region. As you know, mitral murmurs are occasionally transmitted over large areas, sometimes indeed over the entire front of the chest. There is also a soft systolic murmur over the aorta. The cardiac area is not enlarged to percussion. The liver is not palpable. There is no edema of the legs. The urine is normal.

**Diagnosis, Therapy and Prognosis.**—It is apparent that the child has a mitral lesion and that she is at present fully compensated. The history leaves no doubt that she had an attack of heart failure two years ago. The lesion therefore seems now to have reached a quiescent stage. It is possible, however, that the occasional attacks of hemicrania are of rheumatic origin; and that they represent rheumatic recrudescences sufficient to keep up cardiac irritability. You recall that the child's chief complaint was rapid heart action only after rather violent exercise, such as jumping and running. Even normally, of course, there is increased cardiac rapidity after such exertion. Let us see what effect rapid walking across the room has upon the cardiac condition. You observe that there is a rather sudden increase of the

cardiac rate to 110, and that cardiac hyperactivity is now plainly visible, for you see the violent cardiac impulse against the chest wall even from a distance. The child herself, however, does not complain of or notice this hyperactive, increased rapidity. In other words, this girl apparently is conscious of rapid heart action only when it is excessive. This fact is important in determining what she, or other patients for that matter, mean by rapid heart action, for we cannot always examine patients when symptoms or complaints are most obvious or severe. Each patient has his own standard, his own pathological threshold, so to speak, from which he measures the beginning of his complaints. To return to this patient, you observe that the tachycardia is somewhat decreased by deep breathing, a clinical evidence of vagus control, and always an encouraging sign, I believe, for it shows a tendency to normal inhibitory control. Since the hemicrania may be of rheumatic origin, and hence an indication that the rheumatic poison is still active occasionally, I believe the child should take the salicylate of soda in 15 grain doses every night for about a month, and then in decreased doses at longer intervals. While the girl should be allowed to walk about, go up and down stairs slowly and to attend school, I do not think it wise for the present to allow her to romp, nor to do anything which is going to race her heart.

I believe the ultimate prognosis is good. By "good" I mean that the lesion is now quiescent or will become quiescent soon, with no or very little loss of cardiac reserve, with no hyperirritability, and no abnormal tachycardia even upon strenuous exertion. When that stage is reached, the probability is that the mitral murmur will become less loud, and in the course of years may become almost or entirely inaudible.

Case 67.—Mrs. R. N., aged 33, married, has two children, gives no previous history of rheumatism, tonsillitis or scarlet fever. She at no time had cardiac symptoms until the fourth month of her second pregnancy. She then had precordial pains which the family physician diagnosed as due to pleurisy. There was no further trouble during the pregnancy. After the birth of her child, over a year ago, she began to complain of precordial pains which radiated to the left arm and around the chest, and of occasional dry cough. These symptoms were aggravated when climbing stairs and walking fast, or when she became excited and irritable.

**Examination.**—You see that the patient has the appearance of a healthy woman, with good color, well nourished and with no dyspnœa when quiet. Inspection of the chest and neck reveals nothing abnormal. Upon palpation under the left breast, however, you feel the typical presystolic thrill indicative of a mitral stenotic lesion. This diagnosis is readily verified upon auscultation, for you hear the classical diastolic rumble of mitral stenosis, as well as a reduplicated second sound at the apex. Over the pulmonary artery, you hear typical pleuro-pericardial friction sounds especially when the patient breathes deeply. The teeth and tonsils are normal. There is no

edema of the legs. The urine, knee reflexes and abdominal examination reveal nothing abnormal. The lungs are normal.

The orthodiascopic tracing being passed around shows that the pulmonary artery is widened, that the left ventricle is enlarged downward, and that the right auricular curve is somewhat enlarged.

To sum up, the patient has an old quiescent mitral stenotic lesion of which she was not even cognisant until the middle of her second pregnancy, now about one and one-half years ago. She then developed an acute pleuro-pericarditis accompanied by precordial pains. The pains again recurred after the birth of her child. In addition to the pains, exertion and excitement bring on what the patient calls "palpitation." During our entire examination, however, you may have observed that there was no abnormal increase of the cardiac rate so that what the patient characterizes as "palpitation" is probably cardiac hyperactivity.

I have shown you other patients in whom pregnancy or parturition had a definite effect in lighting up dormant quiescent cardiac lesions. I believe that the onset of the acute pleuro-pericarditis was the first tangible evidence of an endocarditic recrudescence in this individual, and that it served to mark the change from the quiescent to a more active cardiac lesion.

**Examination Two Weeks Later.**—You examined this patient two weeks ago. She feels much better now. The treatment consisted in rest for an hour after each meal, very little stair-climbing and a local application of mustard paste over the site of the pain. Internally I prescribed a mixture of salicylate of soda, bromide of soda and simple syrup. The patient complains of very much less pain and "palpitation." Objectively the presystolic murmur and thrill are much less pronounced. This of course does not mean that there has been any recession in the pathological process in the mitral valve, but that the hyperactivity has diminished, with a corresponding diminution in the loudness and harshness of the murmur. In other words, the heart is probably acting now similar to the way it did in its previous quiescent state.

**General Comment.**—I have grouped Cases 82, 83 and 84 because they are examples of irritable hearts in patients with actual cardiac lesions. In two of these hyperexcitability showed itself by tachycardia. In the third, (Case 84), the hyperexcitability was evidenced not by tachycardia, but by overforceful action of the heart which the patient characterized as "palpitation." Case 82, to judge from the history, has apparently been neurotic since childhood; the somnambulism in childhood is evidence of this. Indeed this neurotic taint may be a reason why a comparatively slight and not very active lesion produced such marked tachycardia. Case 83 is an example of hyperexcitability following an inflammatory lesion a year or two previously. Case 84 typifies the results of a local pleuro-pericarditis upon a heart lesion which for years had been inactive.

It is important to know, to study, and to recognize the various kinds of individual reaction when the heart becomes affected. In some, cardiac symp-

toms recede within a few weeks, or after the inflammatory condition has run its course. Thereafter, except for objective changes, cardiac irritability is no longer a symptom, and within certain limitations, the circulation is carried on with no systemic or subjective disturbances. This of course refers to mild endocarditic infections. On the other hand, in patients who are or who become neurotic from any cause, it is the neurotic condition which continues for months or even years after the inflammation of the heart valves becomes quiescent. Indeed in such individuals, any abnormal excitement, let alone any rheumatic or inflammatory recrudescence, can immediately cause a lighting up of all the old symptoms such as tachycardia, precordial pains and dyspnœa. The student must therefore carefully weigh the influence of the neurotic condition itself in such patients, and guide his therapy and prognosis accordingly. I do not wish it to be understood that I consider these patients hysterical; as a matter of fact I have rarely found them so. To state the matter in another fashion, the nervous reflex arcs of these individuals are hypersusceptible, and react violently and continuously to any abnormal stimulus.

Although the pathological process recedes at the same rate in neurotic as in other people with heart disease, the former are "heart sick," so to speak, for a much longer time, the symptoms continue, and they suffer much more than ordinary individuals with similar lesions. Therapy along general neurological lines, therefore, plays a large role with them. The bromides are of value medicinally. These patients are self-centered; therefore it is of great therapeutic importance to divert their minds as much as possible. As soon as the state of the circulation and of the endocarditis allows it, they should be encouraged to seek mental relaxation as well as physical exercise by playing games. Billiards and pool when available, suggest themselves as the mildest at the beginning. Light calisthenics is also an excellent method for diversion as well as exercise. Pitching and throwing ball represents more active exercise. Other amusements and exercises will suggest themselves to fit individual cases and requirements.

### CASE 68.—CONGENITAL PATENT DUCTUS ARTERIOSUS WITH VERY SLIGHT CARDIAC SYMPTOMS

Miss W., aged 22, is the youngest of ten children. She never had rheumatism or any cardiac symptoms until three years ago. She then noticed an occasional "dragging feeling" in the chest, with momentary sharp precordial pains radiating to the back. These pains are not associated with exercise. She has no dyspnœa when dancing, skating or running.

**Examination.**—You note the normal appearance of this young girl; there is no dyspnœa or cyanosis. The pulsations in the neck appear normal. Upon palpation you feel the overacting pulmonary artery in the second left interspace, as well as a somewhat overacting apical impulse. The sounds over the right base and mitral area are normal. Over the pulmonary artery

there is heard a rather rough, loud diastolic murmur transmitted downward along the fourth interspace. This murmur is not influenced by respiration or exercise. It is heard posteriorly between the angle of the scapula and the spinal column; the first and second pulmonic sounds are also heard distinctly in the same area. There are no pericardial friction sounds. The orthodiascopic tracing you are now examining shows that the aorta is normal. The curve of the pulmonary artery is enlarged, and fluoroscopically the artery shows vigorous pulsation. The remainder of the cardiac outline is normal. The abdominal examination reveals nothing of note. The urine and reflexes are normal. There is no edema of the legs.

The diastolic murmur at the left base indicates pulmonary insufficiency; a functional cardio-respiratory murmur frequently found in the same area is but rarely diastolic in time and is not transmitted posteriorly. There are only two valvular lesions which can produce a diastolic murmur over the pulmonary artery: The Graham-Steele, which occasionally accompanies a decompensated mitral regurgitant lesion and is due to relative insufficiency of the pulmonary valve; and a congenital patent ductus arterious with pulmonary insufficiency. In our patient, a Graham-Steele murmur can be excluded because there is no evidence of a mitral lesion. The typical thrill, the systolic murmur, and cyanosis, all characteristic of a marked case of patent ductus arteriosus, are here absent. It is therefore somewhat hazardous to make a positive diagnosis of a congenital anomaly even by a process of exclusion. But it must be remembered that characteristic symptoms and physical signs depend entirely upon the degree of patency of the duct; if it be of comparatively large caliber, the physical signs indicating stenosis may be slight or absent, and the only auscultatory evidence may be that of pulmonary insufficiency from a certain amount of blood regurgitating backward through the pulmonary valves.

The onset of symptoms in this patient—the occasional precordial pains—is difficult to explain. There had been no prior infection to produce endocarditis; there is no evidence of pericarditis. The main danger in very mild and favorable cases such as this lies in the common experience that a patient with any congenital lesion is particularly prone to attacks of endocarditis. Our main therapeutic problem therefore is the prevention of such infection. At present we possess no specific method of guarding against this, except the general rules regarding tonsillitis and rheumatism, and a thorough search for possible foci of infection. Regarding the latter, I am of the opinion that there has been too much interference and that too much radical surgery has been done to remove or drain assumed infected foci, in the absence of any definite correlation between the infection and the cardiac disease.

### CASES 69, 70.—CONGENITAL CARDIAC LESIONS, PROBABLY PATENT INTERVENTRICULAR SEPTUM

I am fortunate in being able to present to you two cases with similar congenital defects.

CASE 69.—T. M., female, aged 9, is the youngest of three children. There is no history of rheumatism, scarlet fever or tonsillitis. She was born a "blue baby." As an infant her face would become quite dusky when she cried. She goes to school now and ranks fairly well in her class. She can walk and even climb stairs slowly without dyspnœa or cardiac discomfort. She has no cough and in fact has no complaint of any kind.

**Examination.**—The child, you see, is well developed. You observe the moderately cyanotic hue of the lips, cheeks and ears. The fingers are moderately clubbed. I want you to note also the interesting congenital anomaly of the right pupil; a triangular sector of it is missing. There is no overaction of the veins or arteries at the root of the neck. Inspection of the precordium reveals no abnormal pulsation. Upon palpation you feel a rough systolic thrill, chiefly over the mitral area. This thrill I want you particularly to observe is not transmitted to the carotids. Upon auscultation you hear a loud rough systolic murmur especially pronounced over, and slightly to the left of, the midsternum; from there it diminishes in intensity, although it can be heard over the entire front of the chest. It is also transmitted slightly posteriorly. The liver is palpable two inches below the free border of the ribs. The lungs are normal. The feet are dusky and the toes are clubbed, as you see. There is no edema of the legs.

I want you carefully to observe with me the heart as I fluoroscope the patient. In the first place you see that the aorta is normal in size, and that there is no aortic overaction. The area occupied by the pulmonary artery, which forms the curve immediately beneath the aorta, is only slightly enlarged. The left ventricle is moderately hypertrophied. The electrocardiogram is of some interest since it shows the characteristics of left ventricular preponderance; namely, a negative ventricular deviation in the third lead; this is contrary to the usual findings in congenital heart disease.

**Diagnosis and Differential Diagnosis.**—The child doubtless has a congenital lesion. There are two principal congenital lesions that can produce marked cyanosis—a patent ductus arteriosus and a patent interventricular septum. In the former the abnormal physical signs—the murmurs and thrills—are especially prominent in the second left interspace over the site of the pulmonary artery and are commonly transmitted to the aorta in the jugulum. Fluoroscopically, there is usually enlargement and marked hyperactivity of the pulmonary artery. On the other hand in those with patent interventricular septa, as exemplified in our present case, the abnormal physical signs are found chiefly over the mitral area, and fluoroscopically, there is commonly no hyperactivity or enlargement of the pulmonary artery or aorta.

CASE 70.—A. S., male, aged 14, is the younger of two children. He has had mumps and chickenpox, but no scarlet fever or rheumatism. When three years old his heart was examined; the mother was then told that the child had congenital heart disease. The tonsils were removed several years ago.

The only symptoms he complains of are rapid heart action and slight dyspnœa when excited or running. He swims and rows without cardiac complaints.

**Examination.**—The boy, you see, is tall and well set up for his age. The systolic blood pressure is 120; the diastolic, 68. In contrast to the other patient, this boy shows no cyanosis of the face or hands. The teeth and pharynx are normal. Upon inspection you observe some hyperactivity of the left ventricle, and of the aorta in the jugulum. Upon palpation you feel a marked thrill over the mitral area. Upon auscultation you hear a loud rough systolic murmur over the same area. This murmur is transmitted chiefly to the left axilla. It is also heard distinctly posteriorly in the upper left interscapular region. It is heard faintly over the aorta in the interclavicular notch as a soft, distant systolic murmur. The abdominal examination reveals nothing of note. The lungs are normal. There is no edema of the legs. The urine is normal. The orthodiascopic tracing, as you observe, shows a normal sized aorta, a somewhat enlarged pulmonary artery and a moderately hypertrophied left ventricle. The right side of the heart is not enlarged.

**Diagnosis.**—As in the previous instance (Case 69) the abnormal physical signs are most prominent over the mitral area and less or almost entirely absent over the aorta and pulmonary artery. Hence my belief that here also we are dealing with a similar lesion—namely, a patent interventricular septum.

**Prognosis.**—Children with congenital lesions come to the physician not because the parents are interested in the type of congenital defect—the parents already know that there is some congenital heart trouble—but because they are interested in the question of longevity; in other words, in what we call prognosis.

I believe the chances that the boy (Case 70) will live at least until adult age, are good for several reasons. (1) His range of activity—his cardiac reserve power—is excellent; you recall he takes active exercise which is not followed by any deleterious effect. (2) He has no cyanosis. This in itself bespeaks a favorable cardiac outlook. (3) He has but *one* congenital defect, as far as we can determine. In those in whom more than one cardiac anomaly is present, the chances for a long life are greatly diminished. To sum up, we may say that thus far the only visible untoward effect of the congenital lesion has been moderate ventricular hypertrophy. This of course is a handicap especially at his age, but it is known that ventricular hypertrophy not combined with inflammatory disturbances may reach immense proportions before the circulation finally becomes severely crippled. And if in this boy the hypertrophy be only slowly progressive the circulation within certain limitations may be carried on fairly satisfactorily for many years. Indeed, a patient soon learns what he can and cannot do with comfort.

A frequent danger in cases with congenital cardiac defects is the possibility of inflammatory valvular infections to which they are especially prone.

Practically this means those individuals should be shielded if possible from rheumatism, tonsillitis and scarlet fever. The boy's tonsils have already been removed. We possess no specific preventative anti-rheumatic measures except the usual ones of care against over-exposure to inclement weather.

The boy may walk and row, and swim in shallow water. He may golf. He should not indulge in the more violent sports as running or tennis playing because of the danger of the development of tachycardia; rapid heart action, once developed, may then become a very annoying symptom and a difficult one to control.

The girl's chances for longevity seem less bright. The cyanosis and clubbed fingers, and the fact that quick walking and stair climbing cause dyspnœa are evidence of a markedly disturbed circulation and of crippled cardiac reserve.

### CASE 71.—TACHYCARDIA AND COLLAPSE AT PARTURITION

Mrs. M., aged 35, is a primipera. The only thing of note in her previous history is that she has occasionally complained of slight "palpitation" when excited or when her stomach was upset. The labor was perfectly normal; there was no inordinate amount of bleeding. The doctor in attendance then followed the usual procedure for the delivery of the placenta, that is, he used the Crédé method of expression. Apparently the placenta was adherent, for even vigorous attempts at expression failed to dislodge it. Within a few minutes after this procedure, the patient became pale and dyspnoic, and the pulse rate ran up to 130 per minute. There was no evidence of any internal or external bleeding. I saw the patient about one hour later. The systolic blood pressure was 60; the diastolic, 45. The cardiac rate was 130 and regular. There was no evidence of any organic cardiac lesion. The facies was pale, the patient was listless although she could be sufficiently aroused to answer questions. She presented a typical picture of circulatory collapse, yet there was no sign of external or internal bleeding. The uterus was well contracted although the placenta was still *in situ*. The placenta was extracted manually one hour later and the uterus packed. Even following this manipulation, the loss of blood was not more than two or three ounces. I gave the patient $\frac{1}{6}$ of grain of morphine, 1 c.c. of a 1–1000 solution of strophanthin, and 1 c.c. of pituritin soon after I examined her. Hot black coffee was given by mouth. These had no effect upon the collapsed condition of the circulation, nor upon the patient's sensorium. Improvement began, but very slowly, only after the placenta was removed. You see the patient now two days after delivery. The temperature is normal. The uterine packing has been removed. The uterus is well contracted. If you listen to the heart, you find the rate around 95. Except for this slight increase in rapidity, the cardiac examination reveals no abnormality The patient is still somewhat pale.

The question of interest is the cause of the circulatory collapse and of the tachycardia. I believe that active hemorrhage, internal or external, can be etiologically excluded because there was no uterine tear nor any other cause for bleeding. It seems to me that the pressure upon the abdomen for the removal of the placenta was the immediate cause of the circulatory collapse. You recall that the history stated that collapse began very soon after this manipulation. Such abdominal pressure can probably produce circulatory failure either by its direct effect upon the venous trunks, or by its influence upon the intra-abdominal ganglia and nerves which control vaso-motor tone. The latter hypothesis seems more probable. For example, you have often heard of, and no doubt know the mechanism of the circulatory collapse following a hard punch in the abdomen. I believe the etiology in our patient was quite similar. The "blow" was the pressure of the hand upon the abdomen in order to express the placenta; the effect was vaso-motor paresis. With over-distended abdominal veins in the splancinic area, or according to some, with over-filled capillaries thus produced, the arterial tree becomes empty, and the patient bleeds into her own veins, or possibly, into her capillaries. The resulting picture is very severe anemia and collapse, as if the patient actually had a severe hemorrhage. Should you ask, why such accidents do not occur more frequently, I can only answer that so many unknown physical factors are concerned in expressing a placenta, that, lacking such data, an answer would be a mere guess; just as we do not understand, and can only surmise why one man is merely stunned by a blow upon the abdomen, and another perhaps killed.

### CASES 72, 73.—TRANSIENT HYPERTENSION WITHOUT ORGANIC CARDIO-VASCULAR DISEASE OR CARDIAC SYMPTOMS

CASE 72.—M. S., female, aged 57, had six children. She reached her menopause six years ago. She has always considered herself "sickly." For years she suffered from hysterical fainting spells and from gastric disturbances; the latter consisted mainly of belching and of intestinal hypermotility. An X-ray examination of the stomach, made some time ago, showed no abnormality. She recently suffered from left sided hemicrania and from blurred vision in the left eye. An oculist's report at that time stated that the patient had glaucoma. The systolic blood pressure then was 175. She recently had two attacks of precordial pain referred to the left axilla. She has always been thin and is a very meager eater.

**Examination.**—You note that the patient is somewhat emaciated. Upon inspection you observe some hyperaction of the carotids in the neck and of the ventricle in the region of the left nipple; this overaction may be only apparent because the neck and chest wall are thin. There is no pain on pressure over the heart. The ventricular overaction is quite noticeable on palpation, in fact, there is a slight thrill-like thrust felt by the examining hand. Upon auscultation you hear a somewhat thrill-like first sound at the apex. When

the patient lies down, however, this abnormality disappears. The other cardiac sounds are normal. The orthodiascopic tracing that I am now passing around shows a somewhat enlarged aortic knob; the remainder of the cardiac outline is normal in contour and size. The systolic blood pressure is now 152; the diastolic, 100. The urine is normal; there is no edema of the legs. The knee reflexes are very lively.

Because of the distinct neurotic history, because of the old attacks of hemicrania, and because there is very little, if any, cardio-vascular disease present, the hypertension seems to be of functional origin and transient in character. This is my belief despite the diagnosis of glaucoma, which as you know is sometimes found in patients with cardio-vascular disease. I do not think that the abnormal sound at the apex indicates organic disease, for it is common in hyperactive irritable hearts. If the glaucoma yields to treatment, I shall allow the patient to continue with her housework and shall put her upon a liberal, fattening diet, consisting chiefly of sweets, starches and fats. We shall have the opportunity of examining her again in a few days.

**Examination Two Weeks Later.**—The physical examination of the heart is the same as we first found it. She has been to another ophthalmologist since we examined her one week ago; he found no evidence of glaucoma nor of any visual disturbance. This does not necessarily mean that the original diagnosis was wrong; the glaucoma may have yielded readily to local treatment or the cure may possibly have been concomitant with the fall of blood pressure, for the latter was 115 and 118 systolic, at the last two examinations.

This case is another instance of the extreme importance of not making a positive diagnosis of *permanent* hypertension from one examination alone —especially if other factors are present which bespeak a marked lability of the vaso-motor mechanism.

CASE 73.—A. D., aged 43, laborer, has been a steady whiskey and beer drinker for years. He has been very "nervous" for several weeks; he cannot sleep, he is unable to work, his hands tremble and he vomits every morning.

**Examination.**—The history, the coated tongue and the trembling fingers which you observe when the patient's hands are outstretched, indicate that he is suffering from alcoholism. The cardio-vascular, the X-ray and urine examination reveals nothing abnormal except the blood pressure, and it is for that reason that I present this patient. The systolic blood pressure is 165; the diastolic, 105. In view of the fact that alcoholism can produce nephritis, the high systolic reading is suspicious. On the other hand, the patient is on the verge of an attack of delirium tremens, and this alone, with its accompanying abnormal nervous state, may also account for the hypertension.

**Examination Two Weeks Later.**—The patient states that he has stopped drinking whiskey; he feels well and has no gastric disturbances. You observe

that his fingers do not now tremble when his hand is outstretched. The systolic blood pressure is 130; the diastolic, 80. The urine is normal. It is therefore evident that the nervous excitement incidental to the incipient delirium tremens was responsible for his temporary hypertension.

### CASE 74.—HYPERTENSION—DIABETES—VASO-MOTOR SYMPTOMS

Mrs. K., aged 68, has hypertension and has consulted various physicians for this symptom for ten years. She says that she has had sugar in her urine, at times as much as 7 per cent. Despite the hypertension, the patient has always been active. For the past few months she has had faint and weak spells at irregular intervals; she has also had occasional "palpitation," dizziness and flushes. There is now precordial distress. Her physician states that while at the seashore, the patient was capable of taking long walks, and had no symptoms. In the city she is quite excitable and is particularly worried because she cannot take active part in the household management; she is always fussing about little household matters. Her physician states that her usual systolic pressure is around 170 m.m.

**Examination.**—You notice the patient is well preserved for her age. You observe also her flushed cheeks. The systolic blood pressure is now 145; the diastolic, 80—nearly normal figures for her age. The radial arteries are not thickened. There is slight aortic overaction in the jugulum. There is no precordial sensitiveness. The cardiac area is normal to percussion. With the exception of a slight blurring of the first sound at the apex the heart sounds are normal. The liver is not palpable. There is no edema of the legs. The urine to-day contains 1½ per cent. of sugar, no acetone or diacetic acid, no casts or albumen.

From the cardiac aspect the patient presents only one definite physical finding—occasional hypertension. Her blood pressure, to-day almost normal, has doubtless been higher at various times. Its variability shows that the vasomotor system is unstable and labile, and is apparently easily affected by various influences. A little nerve upset may readily increase the blood pressure of such individuals. Since there is no evidence of cardiac disease or of cardiac failure, I attribute the patient's symptoms—flushes, giddiness and faint feelings—to the same cause, namely, vasomotor instability. Without a study of the blood sugar, and the effect of diet upon the blood and urinary sugar, it is impossible to state whether this case is one of actual diabetes or whether it is due to changes of vasomotor tone, for the latter may also cause glycosuria. On the other hand, diabetes and arteriosclerosis with hypertension are often concomitant. In our patient, in the absence of any evidence of arteriosclerosis I am not inclined to believe that arteriosclerosis produced diabetes despite her age. As already stated the primary cause of the glycosuria as well as the cause of the hypertension may be instability and lability of the vaso-motor system.

The main therapeutic indication is to get the patient out of her present environment of petty family worries and excitement, and to let her again go to the sea shore, or to any other place away from home. There need be no limitation of exercise. Regarding the glycosuria, the diet for the present should be only moderately restricted. While an advocate of the Allen treatment for most cases of diabetes, I fear that harm would be done in this case by following that treatment now, because the patient would become worried and the symptoms probably intensified. Bromides are indicated: The solid extract of the suprarenal gland in two grain doses should be given for special attacks of weakness and dizziness.

**Later Report.**—The patient's physician told me that she had been out West, where she travelled and walked about without any restriction and with no household cares or responsibilities. She felt well and had no complaints of any sort.

### CASE 75.—HYPERTENSION AT THE MENOPAUSE WITHOUT ORGANIC CARDIO-VASCULAR DISEASE

M. M., aged 53, gives the following history. She has had neither tonsillitis nor rheumatism. She has had six children. Her menopause occurred two years ago. Before the menopause was finally established, she suffered considerably from flushes and perspiration. About 20 years ago she had attacks of severe epigastric pain and vomiting; these attacks often came at night. She has had no attacks of this kind for 10 years. For the past two years she suffered from nausea and pyrosis. Analysis of the stomach test meal shows moderate hyperactivity. She is also enteroptotic, as you will later see for yourselves. The patient was sent to us by her doctor because she has hypertension and extrasystoles; the latter come chiefly upon exertion.

The history is sufficiently complete regarding her gastric complaints. I wish to add, for it is of extreme importance, that the patient herself does not complain of her heart; she has neither dyspnœa, oppression nor any cardiac symptoms.

**Examination.**—The patient, you observe, is not dyspnoic when at rest nor when walking about the room. The systolic blood pressure is 175; after the cuff pressure has been allowed to remain on for a few minutes, it falls to 160; the diastolic pressure is 108. The thyroid gland is not enlarged. The urine is normal. All the heart sounds are normal. There is no precordial sensitiveness nor other abnormality on palpating the cardiac area. When the patient is at rest you hear an occasional premature contraction; their number is somewhat increased by exercise. The electrocardiogram being passed around shows that the extrasystoles are ventricular in origin.

The liver is palpable about one inch below the free border of the ribs. There is slight epigastric sensitiveness to pressure. The orthodiascopic tracing, you observe, shows slight enlargement of the aorta; it is otherwise normal. There is no physical or clinical evidence of cardio-vascular disease

despite the extrasystoles and hypertension. I believe that the gastric symptoms are functional in origin, and that the arrhythmia and hypertension are also of neurogenic nature, caused by nerve instability incidental to the menopause.

Therapeutically, I would suggest atropine sulphate in small doses. This usually has a marked effect upon the gastric symptoms; it may thus help to break the morbid reflex arc now existing between the cardiac and gastric nerves. Appropriate diet for the hyperacidity is also indicated. The patient need not limit her exercise, for the extrasystoles are of functional origin and are but another evidence of abnormal nerve excitation.

Case 76.—H. L., aged 53, has been married many years. The patient is very intelligent and is able to give us interesting and important details not only of her present, but also of her past history. I want to emphasize the following significant data taken from her early history: Since childhood she has been subject to hemorrhages in the tongue and skin. For some years she has suffered from hemicrania and left-sided chest pains; the latter were diagnosed as angina pectoris. Seven years ago a laparotomy was performed for a large ovarian cyst; she has not menstruated since. Hypertension has been present for some years; according to her story, the systolic blood pressure has varied from 225 to 150 m.m. The Wasserman blood reaction is negative. There is an occasional trace of albumin in the urine but no casts. She becomes fatigued very readily. For the last few weeks she sleeps poorly. She knows that her pulse has been irregular for some time; this irregularity is more pronounced of late. She also suffers from occasional attacks of rapid heart action accompanied by dyspnœa. There are no gastric symptoms.

**Examination.**—You observe that the systolic blood pressure is now 210; the diastolic, 130. Upon inspection you note moderate hyperactivity of the ventricle in the apical region, and of the aorta in the jugulum. Palpation over the apex gives a somewhat thrill-like sensation during systole. There is slight precordial pain upon pressure. The abdominal examination reveals nothing abnormal. At present the pulse is rhythmic and of normal rapidity. The urine contains a heavy trace of albumin, and an occasional leucocyte and hyaline cast. The phenolsulphonenaphthalein output in two hours is 60 per cent. There is no edema of the legs. The orthodiascopic tracing as you see shows a somewhat enlarged aorta; it is otherwise normal in outline and size.

**Diagnosis.**—Besides hypertension this patient presents several other features of extreme interest. You recall the early history of submucous and subcutaneous hemorrhages in the tongue and skin respectively, and of hemicrania with precordial pains. Such symptoms have been grouped by Osler under the term "visceral crisis," that is, these individuals are apt to have visceral pains and symptoms especially at the time of the hemorrhages. Often they present no evidence of actual organic disease. The visceral symptoms are sometimes very alarming and almost fulminating in character.

In addition these patients occasionally present marked nervous instability and are especially susceptible to what may be styled "nerve insults." To apply these considerations to our patient, the hemorrhages, hemicrania and precordial pains mark her as particularly prone to react unfavorably to any type of nerve excitation. Hence with the establishment of artificial menopause following the laparotomy, she became a receptive subject, so to speak, for hypertension, tachycardia and extrasystoles—all functional symptoms and signs which one may group as a vaso-motor neurosis.

How shall we evaluate the heavy trace of albumin without casts in the clinical picture? The one phthalein test which was done was normal: The normal excretion for two hours is, as you know, between 40 and 60 per cent. I do not believe we can ascribe the hypertension to nephritic origin, in spite of the albumen. The previous neurotic history and symptoms just alluded to; the absence of dyspnœa and of edema; the absence of gastric and of uremic symptoms all speak against this supposition. On the other hand, I believe it possible that hypertension of the type exhibited by this patient may in the course of time finally eventuate into an actual nephritis. Recurring to the albuminuria, it may possibly be of the so-called orthostatic type, which has recently been shown to be of vasomotor origin in almost every instance. Only more frequent urinary examinations can clear up this point.

Another rather common symptom exhibited by our patient is fatigue: She is "readily exhausted." There are days when such individuals are "all in" and can scarcely walk or get about without feeling unnaturally tired and exhausted. At these times they find it necessary to lounge about and rest mentally as well as physically. Even too much talking then annoys them. They do not care to read or sew. This condition is sometimes precipitated by some untoward, though slight overexertion or excitement. Often, however, it cannot be correlated with such factors. These symptoms are frequently regarded as hysterical, but neither they nor the patients show any of the usual stigmata of hysteria.

Case 77.—S. A., female, 55 years old, had had "bladder trouble" for years until finally relieved by an operation, the nature of which she does not know. She has four children. A hysterectomy was performed upon her seven years ago. Soon thereafter she developed climacteric symptoms—chiefly flushes, and hot and dry feelings over the body. About three years ago her present symptoms began: Dyspnœa of varying severity, pain and pressure over the precordium, and pain along the left arm. Her blood pressure has been high for some time. Occasionally she is awakened by spells of tachycardia which may last an hour or two. She counts her pulse very often. She says it varies from 70 to 100 per minute. She perspires very readily and profusely. She often suffers from attacks of unaccountable fatigue. She has many social activities and does not want to "give in." She says that at times sudden painful swellings of the forearm and wrists appear. Her appetite is good, she has no gastric symptoms.

**Examination.**—The patient who is very intelligent had given me an exhaustive account of her symptoms which I have condensed for you. She is as you see, quite stout. Even now, while she is sitting quietly, you observe that her face is flushed and that she perspires freely. The systolic blood pressure is 210, the diastolic 110. The heart action is regular, the rate, 110 per minute. Upon palpation you will find that there are areas in the third left interspace as well as over the cardiac apex which are extremely sensitive to pressure; otherwise inspection and palpation of the chest disclose nothing abnormal. All the heart sounds are normal, there are no murmurs or accentuations. The lungs appear normal. The abdomen shows the scars of previous operations, otherwise the abdominal examination reveals nothing of note. There is no edema of the legs. The urine and the knee reflexes are normal. The thyroid gland is not enlarged.

The orthodiascopic tracing being passed around shows a normal sized aorta, and a heart which is normal in size and form. The Wasserman blood reaction is negative. The urine and knee reflexes are normal. There is no edema of the legs.

In the absence of physical evidence of cardio-vascular disease, I believe that all the signs and symptoms—hypertension, precordial pains, occasional tachycardia, exhaustion, etc.—are primarily due to the establishment of artificial menopause following pan-hysterectomy. I shall prescribe liberal doses of the mixed bromides, and shall try various extracts of the endocrine glands, chiefly the ovarian and suprarenal, the object being to find a substitute for the lost ovarian secretion.

**Later Reports.**—I saw the patient two weeks later. The systolic blood pressure then was 185, the diastolic, 95. The patient seemed less fatigued but the pains and symptoms were about the same. I made another examination after two weeks. The symptoms were the same; in addition, she had typical gouty arthritis involving a big toe. At my last examination, eight or nine months later, the systolic blood pressure was 175, the diastolic, 110. There was marked skin hyperesthesia over the precordium and epigastrum. The cardiac and other physical signs were the same. A phenolsulphonephthalein test showed an output of 45 per cent. in two hours.

I have as yet found no reason to change my opinion as to the fundamental nature of the case. I believe that both the hypertension and symptoms are of functional origin, and are chiefly if not entirely caused by vaso-motor neurosis following artificial menopause.

Case 78.—N. L., female, aged 49, unmarried, says that she has always been of a nervous and "highly fidgety" temperament. Her menses have been irregular for the past eight years. Two years ago she ceased menstruating. For the last three years she has been suffering from headaches and dyspnœa. She says that her blood pressure has been high for several years. A laboratory report which she has brought with her shows that the urine contains a slight trace of albumen, but no casts, leucocytes or sugar.

**Examination.**—You notice that the eyes of the patient bulge slightly. When the patient extends her hands you observe the coarse tremor which she says she has had for many years. The teeth are in excellent condition; the throat is normal. The thyroid is not palpable. The systolic blood pressure is 200; the diastolic, 135. You observe that there is some hyperactivity of the carotids. Upon palpation you feel a thrill-like systolic shock at the apex. Upon auscultation you hear a somewhat booming first sound at the apex; the second aortic sound is hyperresonant, almost bell-like in quality; the other sounds are normal. The liver is not enlarged. There is a large, readily moveable non-sensitive tumor in the left hypochondrium; it is probably a floating left kidney for it has the general shape of that organ. An enlarged or floating spleen would show the characteristic splenic hilus. The knee reflexes are exaggerated; other reflexes are normal. The phthalein output for two hours is 35 per cent.

The orthodiascopic tracing as you see, shows a slightly enlarged aorta; the remainder of the tracing is normal in size and form.

**Diagnosis.**—I do not think that the abdominal tumor—probably a floating kidney—has any relation to the symptom complex. Again as in the other cases of this group, despite the hypertension, there is very little evidence of any organic cardio-vascular disease. I believe the onset of the menopause is responsible for the hypertension. The somewhat prominent eyes and the coarse tremor are suggestive of hyperthyroidism. Prominent eyes, however, are natural with some individuals, and the patient insists that the tremor is an old one and antedates her present symptoms many years.

I shall allow the patient to do light housework such as cooking, dusting, and light sewing, and advise her for the present to walk slowly. I shall also put her upon a liberal lacto-vegetable diet, and limit her protein intake somewhat. Therapeutically, I shall advise corpus luteum and large doses of the mixed bromides.

**Later Report.**—I have examined this patient several times since I presented her to you. The systolic pressures have varied from 190 to 168, the diastolic from 130 to 120. Once she had slight edema of both legs, but the urine then showed neither albumen nor casts. I prescribed theobromine sodium salicylate in ½ gm. doses four times daily for a few days for the edema, the latter disappeared and has not since returned. Despite the hypertension, the patient is symptomatically improved; she is able to walk better and to do light housework—she complains much less of dyspnœa.

## GENERAL REMARKS UPON HYPERTENSION AND THE MENOPAUSE

Unless a patient belonging to a group such as we have just examined should die of some accident or of an intercurrent disease, and a careful gross and microscopic examination of the cardio-vascular organs be made, there will always be a lurking suspicion, I presume, that the hypertension may not be entirely functional, and that our present methods of examination are not

sufficiently refined or thorough enough to disclose the actual underlying diseased process. I admit the validity of this viewpoint. But unless patients present some of the other common earmarks of actual cardio-vascular disease, I believe that for the present at least, the majority of the hypertensive menopause group must be classed as of functional origin and intimately correlated with the cessation of menstruation.

It must be emphasized that these patients often suffer symptomatically as severely and as continuously, as those with hypertension from actual cardio-renal disease. Thus Case 77 suffers from exquisite precordial pains and hyperalgesia. Dyspnœa is also a common symptom but it is not apt to be as continuous as in hypertension with organic disease. Although the patients complain of shortness of breath, careful examination will sometimes disclose not actual dyspnœa, but rapid respiration, that is, tachypnœa. The latter may possibly be due to hyperirritability of the respiratory center resulting from vaso-motor changes.

Intermittent sweating, sometimes severe enough to be called a sweating attack, is another fairly frequent and annoying symptom of these hypertensive patients. The sweating is sometimes combined with flushes and a feeling of heat in various parts of the body.

The extreme fatigue and nerve exhaustion have already been discussed in conjunction with the presentation of one or two of the cases. I have attempted to discover whether there exists any correlation between the type of onset of the menopause and the onset of hypertension. As you know, with some the cessation of menstruation is slow and gradual; with others, sudden. Some of the climacteric hypertensive cases have suffered from the usual flushes accompanying the menopause, while others have not; so that as far as my observations up to the present have gone, it seems that any patient at the menopause may be a potential subject for hypertension.

The cessation of menstruation undoubtedly causes a profound disturbance of the entire nervous system, and in addition, probably affects some link or links in the endocrine chain. That there exists a more or less intimate correlation between the ductless glands is a fairly established clinical fact, but it is by no means always easy to determine where the chain has been broken, that is, what abnormally functioning gland or glands are responsible for the symptoms. For example, in the hypertensive climacteric group under discussion, it seems probable that the endocrine glands especially affected are the adrenals and the thyroids, as indicated by the hypertension, sweating, tachycardia and the labile vaso-motor mechanism. But it must be remembered that for the present this is only an assumption and not an incontrovertible fact.

The treatment has already been touched upon in presenting the individual cases. Speaking of the entire group, I wish to add that these individuals react only indifferently well to medication. I do not refer to the effect of therapy on the hypertension itself, for I have not found that medication or

diet has any permanent influence on the degree of hypertension. I also made the interesting observation that the patients often feel as well when the blood pressure is excessive as when it is only moderate. I am referring now especially to medication directed to precordial discomfort, dyspnœa and nerve exhaustion. I have found that the extract of suprarenal gland in doses of one to five grains, administered only when such symptoms become most annoying, is followed by excellent results. *A priori*, suprarenal extract seems contraindicated in hypertension but it often acts beneficially nevertheless. The reason for this may be its power to stabilize the vasomotors, thus rendering them less susceptible to extraneous influences. I have only rarely succeeded in giving relief by the administration of other organic extracts such as the ovarian, pituitrin and the thyroid extract. In addition to the suprarenal gland, I frequently prescribe tablets containing atropine sulphate, gr. $\frac{1}{200}$ to $\frac{1}{150}$, and nitroglycerine, gr. $\frac{1}{100}$ to gr. $\frac{1}{25}$, three times daily before meals. This often controls the flushes and sweats.

As for the longevity of the hypertensive climacteric patients, my observation has been that their lives do not seem to be seriously jeopardized, apparently they rarely develop the serious or fatal complications found in the hypertension which accompanies actual cardio-vascular disease, such as coronary infarcts, uremia, pulmonary edema, cerebral hemorrhage, etc.

### CASE 79, 80.—HYPERTENSION AND VASOMOTOR INSTABILITY WITHOUT CARDIO-VASCULAR DISEASE

CASE 79.—This case is quite similar to case 74. C. G., female, aged 69, says that she has had high blood pressure, at times as high as 200, for at least eleven years. She also has had sugar in her urine at various times, even as much as six per cent. During the past few months, she has had "faint-like weak spells" which come at irregular intervals; when they occur she is incapable of doing anything and feels "all in." She also suffers from occasional "palpitation" and dizziness, and from flushes. She has no precordial distress or gastric complaints. While living with her daughter she is always busy with household duties, continually worrying over very trivial matters. When away from the city on a vacation, she is able to take very long walks with no distress, discomfort or attacks of any kind.

**Examination.**—Despite the history of hypertension which her family physician corroborated, the systolic blood pressure is now only 140, the diastolic, 82. The radial arteries do not feel thickened. You note slight aortic overaction in the jugulum. There is no pain or other abnormal sensation upon precordial palpation. There is a slight impurity of the first sound at the apex; all the other sounds are normal. The cardiac area is not enlarged to percussion. The abdominal examination reveals nothing of moment. The urine contains no albumen or casts; it contains one per cent. of sugar, but no acetone or diacetic acid. There is no edema of the legs.

You observe that the patient is not dyspnœic when walking around the room but she does complain of faintness and giddiness. I want you also to remark her extremely flushed face at this moment.

**Diagnosis.**—It is perhaps hazardous to state that this woman nearing her seventieth year, has no arteriosclerosis; yet from the history and from our negative cardio-vascular findings I believe that the hypertension is probably of functional origin. You recall the patient's statement that when away from home and not worried and annoyed with petty household matters, she walks well, has no giddiness and feels quite comfortable. I have no doubt that, as both her physician and she have stated, she has marked hypertension at various times. Indeed the fact that the blood pressure is normal to-day demonstrates the variability and lability of her vaso-motor mechanism. It is perhaps this same instability which causes the glycosuria; for glycosuria, as you know, can be produced by various abnormal nerve influences such as fright, excitement, etc. It will of course require such further examinations as a blood sugar determination and the carbo-hydrate tolerance to test this point.

**Therapy.**—The patient has followed only a moderately rigorous anti-diabetic diet. For the present I shall let her follow her usual dietetic regime. I shall give her the mixed bromides and suprarenal extract, the latter to be taken only when she feels the "weak spells" coming on. I shall advise her to go away from home for several weeks, so as to rid her of the irritation of petty worries. Then she may walk and exercise as much as she chooses.

**Examination and Report Eight Months Later.**—Some of you may recall this patient. She left the city upon my advice and lived with her sister out west for several months. While there, she felt perfectly well, even though she drove and walked around until she was physically tired. The reason she returns to us is that a few days ago, she felt a recurrence of the old symptoms, giddiness, weakness and "palpitation." In addition, she complains of sudden hunger pangs upon arising, and feels as if her stomach were distended with gas. She has pain in the left shoulder and numbness in the left hand. All these symptoms began soon after a sudden attack of diarrhœa which was produced by some indiscretion of diet.

We now find a systolic blood pressure of 160; the diastolic, 78. The cardio-vascular examination is the same as before; there are no murmurs; the urine is normal; the pulse is rhythmical; there is no edema of the legs. The orthodiascopic tracing being passed around corroborates our former finding based upon percussion, namely, a heart normal in size and form.

Why has this patient a recurrence of her old symptoms, in an even more aggravated form? I believe that the patient is now suffering from a gastric neurosis caused by the diarrhœa, and that the former has reflexly excited the vaso-motor mechanism. It demonstrates how hypersusceptible the nervous system of this individual is, that so small a cause can result in such

severe discomfort. I shall attempt to attack the symptoms by treating the gastric neurosis. I shall prescribe atropine sulphate, $\frac{1}{150}$ of a grain, and nitroglycerine $\frac{1}{50}$ of a grain in tablet form to be taken three times daily before meals, I shall also prescribe an antacid powder containing magnesium usta, saccherated peppermint powder and bicarbonate of soda in equal parts, to be taken in $\frac{1}{2}$ teaspoonful doses after meals. Simple semi-solid food should be taken in small quantities about every two or three hours. She is not to wait until her stomach is empty; she will thus avoid the hunger pangs of which she complains.

I have since received several reports from her family physician. She soon began to improve and is at present feeling well, with no discomfort of any kind.

Case 80.—M. H., female, aged 49, presents a symptom complex similar to the previous case, although the history and the course of the disease in the two patients are entirely different.

This patient has had three children. During the last few months her menses have been irregular. For over 10 years she has complained of heaviness in the left arm and of a peculiar "hollow feeling" in the left chest. She is readily fatigued. She had a fainting spell four years ago, the cause of which is not known. Since then she has felt much worse; she has had more precordial pains and can scarcely move without discomfort. Her physican states that the heart action is occasionally irregular and that the pulse rate is usually around 90 per minute. She has never had rheumatism, tonsillar or other infections. At the present time even talking brings on precordial pains and heaviness in the left arm. She has no gastric symptoms. She has complained of insomnia recently. Following the advice of her doctor the patient has remained in bed for several weeks.

**Examination.**—You see that the patient is very well preserved; she scarcely looks her age. Although lying flat in bed you observe that there is no dyspnœa. The systolic blood pressure is 170, the diastolic, 80. The heart rate is 90 per minute and regular. There is a slight systolic thrill on palpation, and a somewhat broken, split first sound on auscultation. As already pointed out such abnormalities are not uncommon in irritable hearts. Otherwise, percussion, inspection, palpation and auscultation reveal nothing abnormal. The lungs, urine, abdominal and neurological examination reveal nothing of note. There is no edema of the legs.

**Diagnosis.**—There is no evidence in this patient of organic cardio-vascular disease; indeed, there is no history of any antecedent infection that might have caused heart disease. Another point against heart disease is the fact that patients with symptoms of actual cardiac disease show almost invariably in the course of a year or two one or more of the usual accompaniments of a cardiac lesion, namely, murmurs, dyspnœa, edema of the legs, etc. From the history, it seems that the symptoms were aggravated by a fainting spell a few years ago. To put the diagnosis briefly, I believe that

she has no organic cardio-vascular disease, but is suffering from an old cardiac neurosis with referred precordial and vaso-motor symptoms.

In this case we base our diagnosis of cardiac neurosis and vaso-motor instability upon the patient's statements and subjective sensations, and upon our negative findings. You may ask, how can we believe in the reality of such symptoms as a "hollow feeling" in the heart? Isn't she hysterical? In answer to that, I wish to state that I have examined many other cases, both functional and organic, who describe their cardiac symptoms in language expressive to them, but which we may not be able to fully comprehend or follow. I may mention for example such descriptions as "empty, tight, gone, fading," "far away" feeling in and about the heart as examples of what patients attempt to convey to the examining physician.

You may also question the patient's statement that talking, or even being talked to, causes precordial symptoms. I have observed just such complaints in other individuals. It is the mental rather than the physical effort involved which seems to irritate these patients and their morbid, hypersusceptible vaso-motor mechanisms.

This patient does not impress me as being hysterical. She is quite calm and is not even worried, although she had been previously told by her doctor that she has "heart disease." On the contrary, she seems very anxious to get better and wants to know what can be done for her.

**Therapy.**—First, medicinally, I shall prescribe some hypnotic to give the patient better nights. Moderate doses of veronal combined with a very large dose of mixed bromides, given every evening, or luminal in one and one half grain doses will best accomplish this purpose. This should be taken for a few nights only. I shall prescribe tablets of the extract of suprarenal gland in three grain doses regularly three times a day, and large doses of nitroglycerine up to $\frac{1}{25}$ of a grain as temporary relief for the precordial symptoms.

Second, the patient will have to be actually retrained in learning first, to sit up in and out of bed, and later to walk about. At the beginning she should err on the side of doing too little; if too much is attempted at the outset, the patient is apt to suffer a relapse, and that is not only discouraging in itself, but it usually requires several days for recuperation. Sitting up in bed, raising the arms and legs slowly *herself*—in other words, active motion—should be the type of exercise at the beginning. Sitting out of bed, standing, and finally walking about the room are the next steps. Thus, by gradations, the patient regains her vaso-motor equilibrium and relearns to walk. All this of course, requires patience and a full understanding of the case on the part of the physician.

Third, Reassurance and encouragement are of vital importance. Without these, other means are of no avail. Such patients are exceedingly apt to have recurrences of symptoms of longer or shorter duration; hence, they must be encouraged over the periods of depression, for they are often depressed when they do not progress steadily. The prognosis regarding longevity is

excellent, that regarding activity is also good, but progress is slow, tedious and usually interrupted by periods of remissions.

**Later Report.**—The family physician reported to me two weeks later that the patient now walks fairly well and has had practically no precordial discomfort.

### CASES 81, 82.—EXTRASYSTOLES WITHOUT ORGANIC HEART DISEASE—COMMENT

CASE 81.—Gentlemen: This woman is 42 years old. She says that the marked kyphosis which you see was due to a fall in infancy. She considered herself well until seven years ago. At that time her child died quite unexpectedly. The shock made her "nervous" and resulted in occasional attacks of "palpitation." On closer questioning I believe we can interpret the "palpitation" as meaning tachycardia. Before her illness the patient weighed 115 pounds; she has since lost about 20 pounds. After two years, the nervous symptoms disappeared and she felt comparatively well until a few months ago. She then developed "stomach trouble," the chief symptoms being anorexia, heart-burn immediately after eating, epigastric distress and sour eructations. There was then neither dyspnœa, palpitation nor precordial distress.

The patient was brought to this Clinic with the diagnosis of heart block. Let us now examine her. We hear no murmurs or other abnormal sounds. Upon percussion the heart seems normal in size. On auscultation we find the following arrhythmia which doubtless was mistaken for heart block: At irregular intervals, either after a few beats or occasionally only after several minutes, a premature beat is heard at the apex, but not felt at the wrist. This type of arrhythmia is frequently termed "missed beat;" when the heart is not auscultated at the same time that the radial is felt, the impression is given that the heart has likewise ceased its activity during this interval. As a matter of fact, what occurs is that the ventricles anticipate their time for rhythmical contraction, and contract prematurely, as evidenced by the premature sound heard at the apex; this sound is fainter than the rhythmic contraction but is still quite distinct. This is another instance of the importance of simultaneous cardiac auscultation and radial palpation. The reason that the premature contraction was not felt as a pulse beat was the purely physical fact that it was not strong enough to open the aortic valves sufficiently to cause a systemic pulse wave and consequent pulse beat. The "missed" or "dropped" beat was missed or dropped as a *pulse* beat, while at the heart, a premature contraction or extrasystole had occurred. I do not care what term you use, so long as you fully comprehend the pathological physiology involved. It is interesting to observe the venous pulsation in this patient's neck, because it is so plainly visible. As you feel the pulse, you observe the normal regular jugular pulsations. With each pulse intermission you note two premature smaller jugular waves which quickly succeed each other;

these represent the waves corresponding to the extrasystole or premature contraction. If a polygraphic tracing were taken, we should find that these smaller premature jugular waves represent a premature auricular, as well as a premature ventricular contraction (the *a'-c'* wave), and constitute evidence of an auricular extrasystole. I wish to add that the patient herself is at no time cognizant of her cardiac irregularity.

Let us now summarize our findings. A frail patient has indigestion for some months. We find she has auricular extrasystoles which vary considerably in the frequency of their occurrence. There is no evidence, physical or otherwise, of organic cardio-vascular disease. How then, shall we explain the arrhythmia? I believe the old history of shock and tachycardia offers us the clue. I have found it to be a clinical fact that once the function of the cardiac nerves is disturbed, the nerves become extremely susceptible to extraneous influence for years, long after the original attack has passed or has been forgotten. I believe the new neurogenic insult, so to speak, is caused by the patient's indigestion, which has again reflexly aroused the old cardiac nerve hypersusceptibility and instability, with consequent production of extrasystoles. You recall that these are auricular extrasystoles, another probable evidence of their functional origin, for it has been found that these are much more frequently of functional than of organic origin.

The therapy should be directed to the stomach, not to the heart. An antacid powder after meals will probably relieve her indigestion. For the extrasystoles, atropine sulphate in doses of $\frac{1}{150}$ to $\frac{1}{100}$ of a grain three times a day before meals may be of value because of its effect in paralyzing the peripheral ends of the vagus. Digitalis is contraindicated because there are no cardiac symptoms nor is there any cardio-vascular disease.

## CASE 82.—VENTRICULAR EXTRASYSTOLES

In conjunction with the previous case (Case 81), I wish to present briefly the following patient. He is 32 years old, has been and is a heavy smoker of cigars and cigarettes. He awoke some weeks ago with a feeling of "palpitation." The next day and often since he has felt as if his heart were "going to stop beating." This feeling, when pronounced, frightens him. Despite these sensations he is able to continue his work, that of chauffeur. He is active and has no dyspnœa upon exertion.

Upon examination we find that there are no cardiac murmurs. There is no decompensation. The heart rhythm is occasionally interrupted by premature contractions which, in contrast to Case 81, cause small, correspondingly premature, radial beats. The jugular veins are not prominent in this individual, hence the study of the jugular pulsations does not help us as before in the differentiation between auricular and ventricular extrasystoles. As you are aware, ventricular extrasystoles can be diagnosed by the presence of *prominent* jugular waves, caused by the coincidence of the auricular and

premature ventricular contraction ($a'$-$c'$ wave). I took an electrocardiogram of this patient; it showed that the extrasystoles were ventricular in type.

To what are the extrasystoles here due? In the absence of cardiac disease and in view of the history of tabagism, I believe that smoking is the cause of the cardiac irregularity.

Student: Is that what we understand by a tobacco heart?

Dr. N.: The term-tobacco heart opens up a very large question. Almost all textbooks state that the poison from tobacco produces very serious pathological lesions in the heart. I have made a special clinical study of a good many cases of so-called "tobacco heart," and I have never been able to find evidence of *organic* heart disease in a single instance. I have found arrhythmias of all kinds: Tachycardias, extrasystoles, and in two individuals, sino-auricular block—but no actual heart disease. As a matter of fact, careful experimental investigations have shown that animals that have been allowed to inhale smoke in an enclosed chamber showed no postmortem evidence of any change in the valves, endocardium or myocardium. This is a contradiction to earlier experimental work done to prove that strong and continued nicotine *injections* could produce aortitis in rabbits. In my opinion, cardiac irregularities following tabagism in man result almost entirely from the effect of the main tobacco alkaloid, nicotine, upon the heart nerves. It is known experimentally that nicotine has a neurotropic effect upon the sympathetic ganglia, and I believe that in the human being, tobacco arrhythmias are caused solely by the toxic effect of nicotine upon the neurogenic control of the heart, and not upon the valvular or other structures. The cause of precordial pain in tabagism I shall not now discuss.

The special reason for showing Cases 81 and 82 was to demonstrate the important fact that the presence of extrasystoles does not necessarily mean heart disease. In one case, tobacco, and in the other, indigestion caused the arrhythmia. Heart disease may of course be accompanied by arrhythmias of various types; but I want you always to remember that arrhythmias in themselves are not evidence of heart disease, and that other symptoms and signs of cardiac disease must be found before you condemn a patient with an arrhythmia as having "heart disease."

### CASE 83.—FUNCTIONAL EXTRASYSTOLES

Mrs. L., aged 49, had always been active and well until the onset of her menopause five months ago. This was at the outbreak of the war when one of her sons entered military service. She identified herself with war activities and worked incessantly from morning until night. Five years ago, the patient had had a mild cholecystitis following gall stone attacks. A few months ago she felt her heart "skipping." She awoke several times during the night with a feeling of "fluttering in the chest." The family physician

states that after a short rest cure the cardiac irregularity became less pronounced.

**Examination.**—You note that the patient is quite robust. She has no dyspnœa. The systolic blood pressure is 118; the diastolic, 70. Inspection of the chest and neck reveals nothing abnormal. There is slight sensitiveness to pressure under the left breast. All the cardiac sounds are normal. The cardiac rhythm is disturbed by occasional extrasystoles. Some of these the patient is aware of and she says that they produce a "weak sensation in the chest." There is at present no sensitiveness in the gall bladder region upon palpation. There is no edema of the legs. The urine is normal. The orthodiascopic tracing shows you that the heart is normal in size and form. Walking, as you observe, does not affect the frequency of the extrasystoles.

I believe that organic cardio-vascular disease can be excluded as a cause for the premature contractions. In a long conversation which I had with the patient before presenting her to you it was made evident that the war and her son's share in it are continually on her mind; these thoughts are further intensified by her own war activities. I believe that it is this nerve strain, occurring at her climacteric which is responsible for the cardiac neurosis and the consequent extrasystoles.

Therapeutically, I shall suggest to the patient that she give up her work for a short while, and then gradually and less ardently resume her activities. To stop her work indefinitely would probably make her very fretful, would result in much more anxiety about her condition, and would give her more opportunity for introspection and worry. When she resumes her activities, she should work in a calm and less hurried way; even in eating, the patient should take her time; for bolting food also has a certain effect in keeping up cardiac irregularities. The mixed bromides should be given every morning. Aromatic spirits of ammonia in teaspoonful doses may be given when she finds the "fluttering in the chest" too annoying. The prognosis depends entirely upon some rearrangement and curtailment of her work, so that her mind and nervous system are not affected by hurry or strain. Indeed the therapeutic problem is a neurological rather than a cardiac one.

## CASE 84.—AORTITIS WITH HYPERTENSION—DECOMPENSATION—EXTRASYSTOLES—NEUROSIS

Mrs. K., aged 66, had always considered herself well physically, although she has been "nervous" of late years. Her nervousness took the form of introspective worry about the possibility of sudden death. Several years ago she was treated for hypertension. She has had two attacks of "grippe." Since the last one she has complained of palpitation and shortness of breath. Her son died about one year ago; this depressed her greatly. She complains frequently of a feeling of "tightness" in her forehead.

**Examination.**—You note that the patient, although of small stature looks well nourished. She is somewhat anemic, yet as she talks her face becomes slightly flushed. The systolic blood pressure to-day is 160; the diastolic, 60. I have taken the blood pressure at various times: The lowest systolic has been 145; the highest, 210. Inspection of the neck reveals moderate overaction of the carotids. Precordial pressure produces no pain. Aortic and ventricular pulsation are normal. Over the right base you hear a loud, rough systolic murmur with a somewhat accentuated second sound. The other cardiac sounds are normal. The cardiac rhythm is interrupted by occasional extrasystoles. These the patient herself recognizes and they no doubt constitute the "palpitation" of which she complains, for the cardiac rate is normal in rapidity. These extrasystoles, taken electrocardiographically, were found to be of ventricular type. The lower border of the liver is one and one half inches below the costal margin. There is slight edema of the legs. The orthodiascopic tracing shows that the aorta is not dilated, and that there is moderate left ventricular enlargement. The urine contains no albumen or casts. The Wassermann blood reaction is negative. The patient's scalp, which she says feels so tight, is smooth, not swollen, and is not sensitive to pressure.

There is no doubt that the patient is suffering from actual cardiac disease: The edema of the legs and the physical signs over the right base are sufficient evidence of that. In addition a marked neurotic element is present. This is evident from the history, as well as from the almost constant complaint of tightness of the scalp. There is considerable lability of the vaso-motor mechanism, as shown by the marked variation in the systolic blood pressure from 145 to 210. Of course, you must remember that a neurotic element can accompany actual disease; indeed, the latter may be the cause of the former. This is especially true of the neuroses which so frequently accompany nephritis with hypertension. I believe, however, that in this patient, the neurosis is simply an added symptom, for with reassurance and local treatment (violet ray was given to the scalp), the tight feeling has entirely disappeared. As I have pointed out in other cases, extrasystoles can accompany and be an evidence of decompensation. And indeed the present examination gives one that impression. But I have examined the patient at other times when there was more tibial edema and hypertension, yet the pulse remained regular and extrasystoles were absent. I believe, therefore, despite the cardio-sclerosis that the extrasystoles are mainly of functional origin. The patient has done well on digitalis and theobromine. Local treatment for the scalp, repeated reassurance—for she frequently harps upon and reiterates her symptoms—have had an important share in aiding this patient. This is another example of the importance of psychology, especially in neurotic individuals. It is always wise to extend hope and sunshine to every patient, and to try to judge of the patient's feelings by the way you might feel under similar circumstances.

## CASES 85–87.—PAROXYSMAL TACHYCARDIA WITHOUT ORGANIC DISEASE—REPORT AND REMARKS UPON TWO OTHER FATAL CASES

Case 85.—P. S., physician, unmarried, aged 34, says that for several years he has been occasionally awakened from sound sleep by a peculiar feeling in his heart. Upon taking his pulse, he always found it regular. He has been a heavy drinker and drinks six to ten glasses of beer daily. During the last few months he has had five attacks of pulse acceleration, the rate at times being 240 per minute. The attacks cease as suddenly as they begin. He has not noted any cardiac irregularity which precedes or ends them. Between paroxysms he has no cardiac symptoms; during them, there is simply an uncomfortable feeling of fluttering in the chest. Three attacks came after heavy meals; belching was then a prominent symptom. After an attack he usually has a slight drawing sensation in the lowermost part of the sternum. During the last few months he has complained of some heaviness in the epigastrium, especially after starchy food. The bowels are regular.

**Examination.**—You note that the patient is rather stout. The systolic blood pressure is 148; the diastolic, 90. The heart action is regular, the rate 100 per minute. The thyroid is not enlarged. Upon inspection you observe that the cardiac impulse is somewhat exaggerated. There is slight accentuation of the first sound at the apex and over the right base; otherwise the heart sounds are normal. There is no abdominal tenderness, no edema of the legs. The urine and reflexes are normal.

The history and the careful observation of the patient himself make the diagnosis of paroxysmal tachycardia unmistakable. I am able to clinch that by the fact that once I observed the patient when his pulse rate was 180 per minute but before I had time to take an electrocardiogram the heart action suddenly became normal.

The question arises, what is the cause of the attacks? Is it to be found in the heart itself or in some other organ? The physical examination of the heart reveals no evidence of disease. The sounds are normal. There is no dyspnœa or other evidence of decompensation. The urine is normal. There is no history of any recent infectious disease. The patient has dyspeptic symptoms which require very careful study because the stomach is often the organ which, when diseased, can excite heart action and bring on tachycardial attacks. I have therefore had a gastric analysis of a test meal and an X-ray examination made. The former shows slight hyperacidity. The only positive finding of the roentgenological examination which included the entire gastro-intestinal canal is slight cardiospasm. This very probably causes the epigastric discomfort, and especially the substernal pain which accompanied the paroxysmal tachycardia. In fact it is probably the cardiospasm which initiates the attack. We know that cardiospasms not due to neoplasms are brought about by abnormal excitation of the sphincteric control of the lower

esophagus and cardia, and that there exists an intimate correlation between the nerves of the stomach and heart. Excitation of one can readily affect the other, although with our present knowledge it is impossible to state why in some individuals, we find reflex excitation of the accelerators and not of the inhibitors. In fact in most instances of gastric disease that I have observed in which the cardiac mechanism was reflexly excited, the heart action has been slowed as the result of vagus excitation, and not accelerated.

Our patient, then, requires treatment for his stomach and not for his heart. He should be advised to refrain from drinking large quantities of fluid of any kind. He should eat moderately and chew his food carefully. He should take an alkaline powder after his meals, and atropine or belladonna before meals. The cardio-spasm and dyspepsia will thus be cured and the tachycardia will probably not recur.

CASE 86.—I. D., female, aged 50, has two children. During the last four months the menses have been irregular. Twenty-two years ago, when her first baby was born, she had an attack of very rapid heart action similar to the present. Since then she has had repetitions about twice yearly. She had a mild attack of typhoid fever seven years ago. There have been no other infections. Three weeks ago, after a heavy meal, she awoke at midnight feeling nauseated. Later she vomited and fainted. When she recovered she complained of rapid heart action which lasted several hours. The family physician found the pulse regular; its rate was 180 per minute.

**Examination.**—The systolic blood pressure is now 150; the diastolic, 90. Upon inspection you note a slightly exaggerated ventricular impulse and slight overaction of the aorta in the jugulum. There is slight pressure sensitiveness over the left breast. The second aortic sound is accentuated, the other sounds are normal. There is slight edema of the legs. The urine report states that there is no albumen or casts and that the specific gravity is 1020.

This patient presents similarities and differences from the previous one (Case 85). Both have had attacks of paroxysmal tachycardia. In both the attacks followed digestive disturbances. In this patient the attack also occurred as the result of acute indigestion. The chief difference is that in contrast to the other, this patient shows some changes suggestive of chronic nephritis: the systolic blood pressure of 150, slight edema of the legs, and the accentuated second sound over the right base. It is now three weeks since the tachycardial attack, hence the rapid heart action in itself could scarcely account for the edema. Upon the probability of presence of a nephritis, even without the urinary changes, I have suggested that the patient be placed upon a salt-free, non-protein diet, that she be given theobromine sodium salicylate in half gramme doses several times daily for a few days, and mixed bromides in 30 grain doses nightly for two or three weeks. The bromides are prescribed to diminish, if possible, the sensitiveness of the nervous system to any abnormal excitation.

Case 87.—L. D., aged 21, as an infant had sudden sharp attacks of vomiting without apparent cause. When six years old she had indefinite attacks of "palpitation." Six years ago she had a mild attack of infantile paralysis. She has had no other infections. During the last two years she has had several attacks of paroxysmal tachycardia; each began and ended with an extrasystole. Such extrasystoles are sometimes called the onset and offset of an attack. In addition to the paroxysms she gets milder spells of rapid heart action; these are usually controlled by taking a drink of water or by resting for a few hours. They often occur after overexercise or when frightened. In the last attack acetone was found in the urine. She has long been on a diet excluding milk, cream, and to lesser extent, sugars. Menses began at 13; they are regular and painless. She has had sudden fainting spells of unknown origin. Her hands are usually cold and moist.

**Examination.**—I had an opportunity to examine the patient during a typical attack of paroxysmal tachycardia which I shall now describe to you. The thyroid was not enlarged. The carotids pulsated regularly and rapidly. On palpation a thrill-like systolic impulse was felt at the apex. Percussion showed that the heart was not enlarged. Upon auscultation a thrill-like, rough, systolic first sound was heard at the apex; there was a reduplicated second sound at the right base and at the apex. The systolic blood pressure was 85; the diastolic, 60. The cardiac rate was 200 per minute and except for occasional extrasystoles it was regular. Pressure upon the right vagus, or holding the breath at the end of a deep inspiration produced a normal pulse rate for a few seconds.

Upon examining the patient now, you note that the cardiac status is normal: The heart sounds are normal, the murmurs that were present during the tachycardial seizures have disappeared. The urine is normal. There is no dyspnœa or edema of the legs. In other words there is no evidence of organic cardio-vascular disease.

I do not believe that acidosis as shown by acetonuria was an etiological factor, because the special diet meant to control it had no effect in decreasing the number of attacks. The cardiac nerve control in this case may be likened to loosely held reins over a skittish, high-strung horse. Any nervous excitement such as hilarity, being startled or even dancing, brings on rapid heart action. When the tachycardia is mild, rest for a short time sometimes stops it; otherwise a severe paroxysm of tachycardia results. The only fundamental cause for the attacks seems to lie in the history of nervous instability of the vasomotor and vomiting centers since childhood.

Therapeutically, I would suggest the extract of suprarenal gland in doses of five grains three times daily for about a month. Although its use is empirical to a great extent, it may act beneficially by stabilizing the vasomotor center. I have found it of value in similar cases. I see no reason for the continuance of the dietary restriction, except of course the avoidance of heavy foods and of over-feeding. The patient also ought avoid those exer-

tions which in her experience have brought on the milder attacks of cardiac rapidity.

Paroxysmal attacks occurring in patients with normal hearts are rarely dangerous to life. The seizure may last several hours, or even several days. In the latter, the tachycardia defies all drug and other therapeutic measures. The seizure may suddenly cease when life is almost despaired of. For the control of the tachycardia, sudden, sharp pressure over the right or left vagus may be tried, by pressing the fingers for a few seconds over the carotids in the middle of the neck. Pressure upon the eyeballs in order to evoke the oculo-cardiac reflex is another procedure which may be of value. Among drugs strophanthin intravenously is occasionally of aid because of its effect upon the vagus. Another procedure is having the patient take a deep breath and holding it as long as possible—this may cause sufficient vagal inhibition to stop the attack (q.v. Chapter XI).

Fatal cases of tachycardia without organic cardiac disease are so rare that they deserve detailed study. I shall therefore offer the following.

## REPORTS AND REMARKS UPON TWO FATAL CASES OF PAROXYSMAL TACHYCARDIA

I recently observed two fatal cases of such sudden arrhythmia—perhaps it would not be correct to term them paroxysmal—which occurred in women after laparotomies.

Mrs. P., 30 years old, said that for some years she had had occasional spells of "weak heart," especially when her stomach was upset. Careful questioning elicited the important fact that by "weak heart" she meant that her heart would suddenly beat very fast, and then would as suddenly resume its normal rate. For several weeks she was in bed, suffering from a right-sided pyosalpinx with irregular fever. A laparotomy was advised. Examination of the heart shortly before operation revealed no murmurs or arrhythmia. During narcosis there was no vomiting but the cardiac rate reached 120 per minute. Narcosis was stopped before the operation—salpingectomy—was completed, because of the rapid heart action. After the operation, the heart remained regular, the rate 160 per minute, the pulse scarcely palpable. When I saw the patient 48 hours afterwards, she was lying flat in bed without any dyspnœa. The cardiac area was not enlarged to percussion, there were no murmurs. The thyroid was not enlarged. The heart rate was 180 per minute; the action, regular. The lips were somewhat cyanotic. The lungs were normal. The hands were cold, the pulse scarcely perceptible. The patient regurgitated fluid frequently, although there were no signs of peritonitis. The rectal temperature was 100°. The mentality was clouded although repeated questioning elicited an intelligent response. The diagnosis of paroxysmal tachycardia without organic cardio-vascular disease was made. First right and than left vagus pressure in the neck was tried,

but the procedure had no effect upon the cardiac rate. One cubic centimeter of $\frac{1}{1000}$ solution of strophanthin and $\frac{1}{4}$ of a grain of morphine sulphate were injected subcutaneously; they also had no effect on the tachycardia. The patient died one hour later. The cause of the tachycardia will be discussed in conjunction with the following similar case.

Mrs. M., aged 35, gave a definite history of hysteria. She frequently complained of her heart during some of these hysterical attacks. She suffered from diseased adnexa which required operation. Examination of the heart at the surgeon's office several days before operation revealed a cardiac rate of 120 per minute; there were no other abnormal findings. The patient was very much frightened at the thought of a laparotomy. The heart was rapid as she got on the operating table. During the narcosis, the patient became somewhat cyanotic. Suddenly, about twenty minutes before the completion of the operation the patient had a convulsive seizure lasting about five minutes. Immediately thereafter the heart rate was 180 per minute, the pulse scarcely palpable. The rate remained the same until I saw the patient two days later. The temperature was 103°; she passed 1900 c.c. of urine the first day after operation. The patient was talkative and answered questions incoherently. The abdomen was ballooned and slightly tense, but not sensitive to pressure. The tongue was coated and dry. There was no vomiting. The leucocytes were 10,000 per cubic centimeter, the differential count was normal. The cardiac rate was 160 per minute, the action regular. There was a systolic murmur at the apex, common to patients with rapid heart action. The systolic blood pressure was 75; the diastolic, 65. Despite warm rectal Murphy drips of glucose solutions, vagus pressure, digitalis in large doses, and adrenalin chloride subcutaneously, the patient died within 24 hours after my examination.

My first impression was that the patient was suffering from sepsis; but the onset of the violent tachycardia during narcosis, ushered in as it was by a convulsive seizure, apparently indicates that in some manner the tachycardia was the fundamental cause of the other symptoms.

Regarded mechanically, sudden overloading of the large deep abdominal veins with blood causes lack of blood to the systemic arteries, and hence to the heart. The heart is therefore never sufficiently filled with blood, and even when beating rapidly, as in tachycardia, there is insufficient arterial blood pumped through the systemic circulation. In itself, this may conceivably account for various abdominal symptoms and for improper functioning of the viscera because of lack of arterial blood. Indeed both fatal cases may be compared with some phases of shock as seen on the battlefield: The low systolic blood pressure, the rapid heart action, the assumed overfilling of the venous trunks and the ineffective ventricular systoles resemble those shock cases in which there is an abnormal distribution of the blood volume, the preponderant amount being found in the veins of the splanchnic area or possibly in the capillary bed.

It would be of great importance to know the exact cause of the inception of rapid heart action in these fatal cases. With our present incomplete knowledge the discussion must be perforce hypothetical. Both cases gave histories of attacks of tachycardia prior to operation. It is of course not unusual to find patients, especially nervous women, with tachycardia induced by the fright of operation. In the great majority of these the narcosis, by dulling sensibilities and (speaking physiologically) by diminishing the threshold of the higher cerebral centers to reflexes, tends to make the cardiac rate resume its usual pre-operative level. In both of these fatal cases, nothing in the operations themselves could have affected the heart rate; both operations were skilfully performed, there was no undue loss of blood nor any especially forceful intra-peritoneal manipulations. In fact in both tachycardia occurred before the operations were completed. Narcosis itself often produces cyanosis, but unless extreme, it rarely causes tachycardia. However it is impossible to state the effect of even slight cyanosis upon sensitive cardio-inhibitory centers already functioning improperly.

Although the question of shock in its many manifestations is still unsettled, its frequent and most damaging effect is sudden overdistension of the abdominal reservoirs; a bleeding into a patient's own veins, with all the serious consequences of an actual external hemorrhage. Another view is that the blood stagnates in the capillaries. It is in some such aspect, I believe that these fatal cases must be conceived. The laparotomy may have been the culminating factor by producing changes in the venous circulation of the abdominal viscera. That tachycardia, once established, can become persistent even after the original cause has ceased, is evident from Cases 85 and 86. In one of these as you recall, tachycardia continued for hours after the original gastric attack had ceased.

### CASE 88.—PAROXYSMAL TACHYCARDIA DUE TO GASTRIC NEUROSIS AT THE MENOPAUSE

Mrs. S., aged 52, passed her menopause three years ago. Since then she has had attacks of severe left-sided hemicrania and rapid heart action. From the patient's description the latter seems to have been typical paroxysmal tachycardia. These seizures usually occurred when she was hungry, several hours after an early breakfast. There are no gastric symptoms. The patient is constipated, sleeps very poorly, and feels so fatigued that she must go to bed early. She usually awakes at about one o'clock with a start, and then is awake the remainder of the night. She is out of bed at six. She has not lost in weight.

**Examination.**—You note that the patient is well nourished. There is no dyspnœa. The systolic blood pressure is 110; the diastolic, 70. Cardiac examination reveals nothing abnormal. The reflexes and urine are normal; there is no edema of the legs. The orthodiascopic tracing, as you see, shows

that the aorta is normal and that the ventricle lies somewhat flat on the diaphragm.

There is evidently no actual cardio-vascular disease to account for the attacks of paroxysmal tachycardia. I believe that the cause of the latter is to be found in the sharp hunger attacks from which the patient suffers every morning. Although I shall not enter at any length into the subject of the sensations of hunger, you know they are the result of nerve influence, probably from the vagus upon gastric secretion and motility. In this simple phenomenon lies the etiology of this patient's tachycardia, the cardiac accelerators become reflexly excited with consequent production of tachycardia. The fundamental cause of the nerve instability is probably the climacteric, which may also be the cause of this patient's insomnia, exhaustion and hemicrania.

The patient must take more rest and sleep. The stomach should not be allowed to become empty. Abnormally hungry feelings should be guarded against by frequent small feedings; milk answers this purpose very well. The patient may continue with her housework. She should lie down for one hour after lunch. Such simple measures will probably cure the patient of her paroxysms of rapid heart action.

## CASE 89.—DISCUSSION OF AURICULAR FIBRILLATION WITH AN ILLUSTRATIVE CASE

This man is 45 years of age. The previous history states that at the age of three, he had some bone infection in his left knee which resulted in an operation and subsequent fistula. There is no history of any other infection —no growing pains, no scarlet fever, no rheumatism, no sore throats except one very mild one which lasted a day or so. He gives a moderate alcoholic history. There is nothing otherwise in the history which has any relation to his present trouble. The Wassermann blood reaction is negative.

The complaint which brings him to the hospital began rather acutely about five months ago. He then became short of breath; he could not walk; his abdomen began to swell.

Let us first study the heart action. Please listen to the heart, not for murmurs at present, but with the primary idea of telling me what you think of his arrhythmia.

Student: There is very rapid, irregular heart action; the first sound seems to me to be very strong.

Dr. N.: For the present I am interested in the rhythm alone.

1st Student: The rhythm is irregular, rapid, and at times seems to be skipping.

2nd Student: His heart is very irregular; first it is slow, then rapid, then it skips.

3rd Student: It seems paroxysmal.

Dr. N.: I notice, in the first place, that none of you gentlemen felt the patient's pulse; that in itself is a very grave error. Every time you examine a heart, particularly when studying and looking for arrhythmias, always feel the pulse at the same time. If you do that in this case, you will notice not only that the heart action is irregular but that the pulse is also irregular; furthermore with every heart beat there is a pulse beat; that is, the pulse is just as irregular as the ventricular action. You will further note that not only is there an irregularity in time but also in the strength of the ventricular beats. There are scarcely two successive beats of equal force or rhythm.

The type of arrhythmia which produces such gross irregularity of force and rhythm of the heart and a corresponding irregularity of the pulse is called auricular fibrillation.

There are various experimental methods by which auricular fibrillation can be produced. A common method is to faradize the auricles directly. As a result, instead of having the physiological orderly rhythmical auricular contractions followed by regular systolic contractions of the ventricles, there results irregular discordant auricular activity. Different parts of the auricle contract at different times and with varying intensity; indeed, as the technical term denotes, the auricles actually fibrillate. At the same time the auricles are in a state of dilatation. What is the result upon the ventricular activity? In the normal rhythmical heart as you know the impulse starts at the sino-auricular node, the so-called pacemaker, and spreads thence through the auricles to the auriculo-ventricular conduction system consisting of the auriculo-ventricular node, and of the bundle of His with its branches. The ventricles are thus incited to rhythmic orderly contraction. In auricular fibrillation, there is as it were a bombardment of the auriculo-ventricular conduction system by a great number of discordant auricular impulses. As many as 900 of these per minute have been counted in the electrocardiograms of patients with auricular fibrillation. Comparatively few of these impulses can pass through the conduction system. Those which do pass incite the ventricle to irregular, tumultuous activity. That is true in the case we are studying. Indeed because of this man's thin chest wall and enlarged ventricles, you may actually see that the ventricular contractions are irregular in strength and rhythm. One may even venture a diagnosis of auricular fibrillation by mere careful inspection of the cardiac area. Please remember that you never see or hear the auricles fibrillate but that you make the diagnosis of auricular fibrillation by the characteristic effect of the latter upon ventricular activity.

What is the result of this irregular ventricular activity upon the systemic circulation? Some of the ventricular contractions are so weak that they cannot open the aortic valves and therefore cannot produce corresponding pulse waves in the radials. Another reason for the non-opening of the aortic valves may be that the ventricular systoles, otherwise sufficiently strong, do not properly direct the blood toward the aortic valves. In either case, it is apparent that there will be more heart than pulse beats. The consequent

lack of correspondence between the number of ventricular systoles and the pulse rate is sometimes called pulse deficit. I prefer the more descriptive name of abortive beats. The stronger ventricular systoles open the aortic valves in the usual fashion and produce correspondingly strong pulse beats. But since even these effective ventricular systoles vary in force and rhythm there is a corresponding variation in the propagated arterial waves as shown by a pulse which is also irregular in force and rhythm.

Let us now verify all these observations by again examining the patient. You will note that some ventricular systoles are followed by pulse beats, while others are not. I wish to impress upon you again that this simply results mechanically from the fact that some of the ventricular systoles do not open the aortic valves.

Why do patients with auricular fibrillation so frequently decompensate? It has been shown experimentally that the reason depends primarily upon the resultant irregular ventricular activity. When the latter is present the blood cannot be thrown into the systemic circulation in an orderly fashion, and it is this which finally produces what we call heart failure or decompensation in patients with this arrhythmia.

Clinically, the incidence of auricular fibrillation may be roughly grouped as occurring, first, in *rheumatic endocarditis;* second, in *cardio-sclerosis;* and third, in patients *without cardiac disease*—the extracardiac group. All these groups will be discussed in detail in succeeding Clinics. Here I wish to state briefly that the rheumatic valvular lesion most apt to produce auricular fibrillation is mitral stenosis. Further, that by cardio-sclerosis I mean a generic term which includes gross pathological changes in all the cardiac structures—valves, endocardium, muscle, aorta and coronaries. It occurs chiefly in older individuals with general arteriosclerosis, with or without hypertension. The third group—those without actual disease—is the smallest in number.

Auricular fibrillation comprise approximately 70 per cent. of all arrhythmias, hence the importance of understanding and diagnosing it. It will also be much easier for you to grasp and comprehend the others if you thoroughly recognize this irregularity. Another practical reason why it is so important to grasp the fundamentals of auricular fibrillation is that it is the one which may most often be benefitted therapeutically. We know that digitalis has the power of blocking many of the auricular discordant impulses which bombard the auriculo-ventricular conduction system in auricular fibrillation. These impulses are thus to a great extent cut off, so that the ventricles are restored to some condition of orderliness in rhythm and force. In addition digitalis also helps by increasing the contractile power of the heart. The great advantage of digitalis, however, in fibrillation arises from the aforesaid power to block the manifold auricular impulses, thereby producing more regular heart action. Quinidine has the remarkable power of occasionally restoring the normal rhythm in some cases of auricular fibrillation. I shall later present some cases to demonstrate its action to you.

Thus far we have concerned ourselves only with the general diagnosis of and the problems arising from the arrhythmia itself. I should now like to have you listen to the heart with a view to distinguishing the abnormal sounds you hear.

1st Student: The sounds are accentuated; there is a murmur.

2nd Student: The first sound is replaced by a loud murmur; a systolic one. I do not think the second sound is accentuated.

3rd Student: There is a murmur which seems presystolic, and at the same time one hears a sharp first sound. The second sound is accentuated.

Dr. N.: Upon auscultation, I find as follows: In an area surrounding the left nipple I hear a rather loud systolic murmur. A little lower and near the cardiac apex I hear a somewhat distant diastolic murmur which occupies the latter half of the diastolic period. Will you please listen now in order to corroborate these auscultatory findings?

The patient's heart action was so rapid when first admitted to the hospital and before putting him on digitalis, that no murmur could be distinguished. But as I have just pointed out we now hear in the left nipple region a rather loud systolic murmur, and in the apical region, a relatively distant diastolic murmur of a somewhat rumbling character. In the presence of auricular fibrillation they are typical of a double mitral lesion: The systolic, of mitral regurgitation; the diastolic, of mitral stenosis.

And now as to the size of the heart. Even without percussion you note the size of the organ as it is silhouetted against the chest wall. The apex is in the fifth interspace in the axillary line.

The liver is enlarged; its lower border can be felt at the umbilicus. There is very slight edema at the ankles.

Let us now search the history for the cause of the valvular disease in this patient. The great majority of cases of mitral disease is caused by rheumatism, tonsillitis or scarlet fever. The patient says he had a slight attack of sore throat some months ago shortly before his cardiac symptoms began. Such widespread cardiac mischief, however, could not possibly have been produced by so recent and mild an attack. It is possible that this patient represents one of those in whose childhood there had been so-called "growing pains"—in other words, rheumatism— and that the patient does not recall them. Or the old bone infection in childhood may be the cause of this mitral lesion. Under such circumstances however, patients are not apt to have such exceedingly long quiescent periods for, as you remember from the history, this man until recently never had any cardiac symptoms. Indeed, he had never consulted a doctor about his heart until a few months ago, a very unusual story of an endocarditis resulting from a septic bone infection. With reference to the alcoholic history in this case, he has been only a moderate drinker. The role of alcoholism as a cause of endocarditis has been, as we now know, overemphasized; and, even conceding alcoholism as an etiological factor, it is difficult to conceive it as producing such a typical mitral valvulitis.

Weighing all probabilities it seems most likely that the cause of the lesion is rheumatism, the details of which the patient has forgotten.

This patient has received altogether a little over two drachms of the tincture of digitalis. This has already produced a very marked change in his condition. The heart beats less frequently and there is a greater tendency to regularity. The edema of the ankles is less; the liver is smaller. The man can breathe more comfortably, and as he says he "feels fine." He would scarcely have to tell us that because a cardiac patient who breathes as comfortably as he does always "feels fine." I am now going to put him upon rather large doses of digitalis, a half teaspoonful three times a day for about three days, and then for the following two days, upon theobromin sodium salicylate with a limited fluid intake.

Regarding further care of this and similar cases, I should advise that after compensation has been restored, the patient consult a physician about every two or three weeks. We know that, given properly, digitalis may be taken for a number of years. This patient may be made so comfortable that he may even be able to follow some light pursuit. Some become so accustomed to taking digitalis that they themselves are able to approximate the proper amount of the drug needed. The average amount of the tincture of digitalis required to restore compensation is one ounce. I am in the habit of giving one drachm a day in the usual mild case of decompensation with auricular fibrillation. In more urgent cases I prescribe much larger doses.

Student: Does the pulse rate influence you in the amount of digitalis to be given?

Dr. N.: If I do not get the proper effect, that is, the restoration of compensation, I would continue digitalis up to the one ounce amount, even though the ventricular rate were to fall as low as 45 per minute. As a matter of fact in order to investigate that point for myself, I had under observation some three or four years ago, a series of cases of fibrillation in whom I continued digitalis despite the fact that the ventricular rate was between 40 and 50 per minute. One patient told me he had never felt better in his life: He left the hospital with a heart rate of 35 per minute.

Student: How often would a case of this sort have to be seen in private practice?

Dr. N.: Ordinarily once daily while in the acute stage. As the patient's condition improves, say at the end of four or five days, he may be visited less frequently.

Student: Do you give tincture alone?

Dr. N.: I always give the tincture alone and undiluted. You can keep a good tincture several months without detericration.

Student: How long do you keep such a patient in bed?

Dr. N.: That depends on the severity of the case. This particular patient will probably improve sufficiently in about ten days so that he will then be able to sit out of bed, and in a few days thereafter, walk around.

Student: Does he feel very weak?

Dr. N.: The usual complaint is shortness of breath. The weakness is not more marked than that of other bed ridden patients. If I allow this patient to get up and walk around without continuing digitalis in smaller doses, he will again decompensate in a comparatively short time, probably in several weeks. Incidentally, what I have just said proves to you that the old theory of the danger of continuing digitalis when a patient is walking about is an erroneous and exploded one.

### GASES 90-92.—DISCUSSION, DESCRIPTION, AND TABULATION OF VARIOUS TYPES OF AURICULAR FIBRILLATION WITH ILLUSTRATIVE CASES

To-day I shall continue the subject of auricular fibrillation and shall illustrate it with several additional cases.

CASE 90.—This patient has already been examined by some of you. M. H. is a typical example of a decompensated double mitral lesion with auricular fibrillation. You will recall that I advised the continuation of medication even after compensation was restored. He has not taken any digitalis since he left here three weeks ago; he re-entered the hospital several days ago, decompensated and dyspnœic. Two or three days after the second admission he developed a left-sided hemiplegia; you see he cannot raise his left arm or his left leg. Upon listening to his heart you hear, just beneath the left nipple, a loud systolic and a distinct, though somewhat distant diastolic rumble followed by a reduplicated second sound. The pulse and heart action are grossly irregular in force and rhythm. The liver is enlarged; there is no edema of the legs. This case, therefore, represents auricular fibrillation with a double mitral lesion probably due to an old rheumatic infection.

The prognosis is now complicated by the hemiplegia, which no doubt is due to an embolic infarct from one of the mitral vegetations. The point I wish to emphasize at this juncture is that the patient did not follow my directions regarding digitalis. I had stated that despite the fact that he was thoroughly compensated when he left the hospital, he would have to continue medication. This the patient did not do, and I am therefore not surprised to find that he returned very much decompensated. It is indeed probable that the irregular violent ventricular action such as is even now present was the controlling mechanical factor which loosened one of the mitral vegetations. If the heart had been beating more quietly and less irregularly under the influence of digitalis, an embolic infarct would have been less likely to occur.

CASE 91.—A. B., male, 76 years old, came to the hospital for a skin disease which has no connection with his cardiac status. I wish to demonstrate him as illustrating a cardiac type very frequently found among the old and senile. He has emphysema and he also has a very irregular pulse. I want you to feel his pulse and to listen to the heart at the same time, a procedure always to be followed when studying arrhythmias. You will find that unless the

patient sits up, you can scarcely feel the heart beat. The sounds are very faint, probably because the organ is covered by emphasematous lung. The radial and temporal arteries are thickened. You note that the pulse is irregular both in rhythm and force, an almost infallible sign of auricular fibrillation. The underlying pathological lesion is cardio-sclerosis, a very frequent finding in elderly individuals. Very often such patients have no cardiac symptoms. In this case, for example, despite the fact that the pulse is grossly irregular, there are absolutely no cardio-vascular symptoms—no dyspnœa, no edema, no decompensation.

Case 92.—This man, 53 years old, a tailor, tells us that except for very slight, temporary ailments, he had never been ill until about five years ago. At that time he sought admission to another hospital and stayed there for two weeks. The symptoms then were shortness of breath and cough. He left the hospital and soon thereafter noticed that his eyes began to bulge, although he felt absolutely well and was able to continue at work for several years. He then sought re-admission to the same hospital because of the recurrence of the original symptoms. After several weeks he left and was again well until about six weeks ago. At that time he developed diarrhœa and shortness of breath. The latter has continued up to admission to this hospital five days ago.

Let us now examine the patient. First, by inspection you note that the patient is very dyspnœic and that he has very marked exophthalmos. You further note very prominent and irregular pulsations in the neck; the larger and more vigorous are due to the carotids. The jugular pulsations are irregular but less prominent. You likewise observe that there is a fullness of the neck in the region of the thyroid; both lobes of this gland are considerably enlarged. In the chest you observe that the apical impulse is diffuse and is very rapid and irregular in the time and force of its systoles.

Second, Palpation: By placing the palm of the hand over the precordium you are able to feel and corroborate what you have just observed by inspection; namely, the diffuse irregular impulse produced by ventricular systoles which are irregular in rhythm and force. In other words the ventricular action is grossly arrhythmic. No thrill is felt over the precordium.

Third, Auscultation: We hear no cardiac murmurs but we do hear some moist, crepitant rales probably due to congestion and edema of the left lung. You also note that some of the ventricular beats do not reach the wrists. That is due to the fact, already pointed out in a previous Clinic, that some of the systoles are not strong enough to open the aortic valves or that they do not throw the blood in a proper direction towards the aortic valves. Either reason is sufficient to prevent opening of the latter and hence prevents a corresponding pulse wave.

Finally, The size of the heart: The apical impulse is most prominent in the sixth interspace, twelve centimeters from the midsternal line, a point near the anterior axillary line; this is evidence of enlagement.

The liver is considerably enlarged; its lower border is two inches below the umbilicus. There is marked edema of the legs. The urine shows hyaline and granular casts. The Wassermann blood test is negative. Regarding therapeusis, I wish to mention that, despite digitalis, morphine and bromides, all given in large doses, there has been no improvement in the patient's condition.

This case is one of auricular fibrillation as shown by the absolutely irregular ventricular action. Because of the exophthalmos, the enlargement of the thyroid, the absence of any rheumatic or infectious history, the sharp attack of diarrhœa antedating the present illness, and because the patient had several attacks of decompensation, I believe that the auricular fibrillation and consequent heart failure are due to exophthalmic goiter ("Basedow's disease"). I shall shortly explain my reasons for this view.

I ask you now to study this tabulation with me, in which I divide the subject of auricular fibrillation as follows:

TABLE I

A. *Experimental causes of auricular fibrillation.*
   1. Stimulation of the vagus.
   2. Stimulation of the sympathetic.
   3. Direct faradization of the auricles.
   4. Injection of thyroid extract intravenously (1 experiment).

TABLE II

B. *Some abnormal factors found in or productive of auricular fibrillation in the human being.*
   1. Enlarged and dilated auricles.
   2. Pathological changes in the pacemaker (sino-auricular node).
   3. Changes in nerve tone in the pacemaker.
      (*a*) From destruction of nerve fibrils and ganglia.
      (*b*) From abnormal impulses reaching the pacemaker.

TABLE III

C. *Auricular fibrillation is found clinically in:*
   1. Cardio-vascular disease.
      (*a*) Valvular disease—chiefly mitral stenosis.
      (*b*) Cardio-vascular—chiefly senile (in both, auricular fibrillation is usually permanent).
   2. Extra-cardiac causes in patients with normal and abnormal hearts.
      (*a*) Toxins.
         Biological—chiefly in pneumonia (auricular fibrillation usually temporary).
         Chemical—tobacco, sulphuretted hydrogen (isolated observations).
      (*b*) Reflex—chiefly, from gastro-intestinal disease (auricular fibrillation usually temporary).
      (*c*) Exophthalmic goiter (A. F.[1] usually in attacks or permanent).
      (*d*) Drugs—chiefly digitalis (A. F.[1] usually temporary).
      (*e*) Emotions—fright, fear, excitement (A. F.[1] in attacks).
      (*f*) As part of, or during attacks of paroxysmal auricular tachycardia.

[1] A. F. = auricular fibrillation.

I assume that you know what I mean by the pacemaker, the normal rhythm center or the sino-auricular node—all interchangeable terms. You know that its arterial supply is a rich one, the node being supplied by its own special artery. It is also rich in nerve ganglia and fibrils.

I want to stress the importance of the knowledge we now possess, that fibrillation may be temporary or may appear in attacks. The older writers regarded auricular fibrillation as a perpetual arrhythmia.

You of course observe that Cases 90, 91, and 92, all fibrillators, belong in different etiological categories. I shall briefly discuss some of the clinical conditions in which auricular fibrillation is found.

Mitral stenosis is one type of valvular disease in which fibrillation is quite common. Usually in decompensated mitral stenosis, where fibrillation has appeared for the first time, the ventricular action is quite irregular, and the rate, 100 per minute or more. Decompensation, as I have already emphasized in a previous Clinic is brought about by the irregular ventricular action which results from fibrillation, and it is the irregular ventricular action which is of such extreme practical importance. That is to say, the thing which disturbs the patient clinically, which causes the dyspnœa, the anasarca, the bronchitis, etc., is the irregular ventricular activity which prevents the blood from being thrown into the general systemic circulation with any degree of regularity.

Although I use the term senile cardio-sclerosis, it must be understood that this pathological type is by no means infrequent in the earlier decades of life. Senile cardio-sclerosis is also frequently accompanied by auricular fibrillation. It is uncommon to have, as in Case 91, rather irregular heart action with rates which do not exceed the normal ventricular rate. The pulse and cardiac irregularity, if you will recall the examination of Case 91, is not nearly as marked in rhythm or force as, for example, in Case 92, the patient with the exophthalmic goiter. That probably accounts for the fact that patients such as the former do not often suffer from much disturbance in the systemic circulation despite fibrillation. Case 91, indeed, came to the hospital for an entirely different disease; the irregular pulse was only an incidental finding. There are, however, senile cardio-sclerotic cases which do have very irregular heart action with fibrillation, and those are the patients who suffer most from dyspnœa and other signs of decompensatory disturbances.

We shall now discuss, in conjunction with Case 92, the question of exophthalmic goiter with auricular fibrillation. By correlating the tabulated experimental (Table I), with the probable fundamental causes of fibrillation (Table II), we may be able to find the cause of the fibrillation in Case 92. I want to preface, however, that what I am going to tell you about fibrillation in exophthalmic goiter is to some extent hypothetical, because, though we have some experimental data, the facts are not sufficiently known to enable us to draw exact clinical conclusions.

You note from the Table that, experimentally, stimulation of the sympathetics has produced auricular fibrillation, and clinically, that changes in the rhythm center from abnormal impulses reaching it may also produce fibrillation. We know that exophthalmic goiter is sometimes accompanied by mild pathological changes in the myocardium; in rare and exceptional instances these may amount to extreme cardiac hypertrophy without involvement of valvular or arterial structures. Furthermore exophthalmic goiter is almost invariably accompanied by evidence of hyperexcitation of the sympathetic system. Observe, for example, the usual tachycardia and sweating in exophthalmic goiter. We know, also, that there are undoubted instances in which sudden emotions such as fright or excitement, form the starting points of exophthalmic goiter. Case 92 states that preceding his last attack of cardiac failure, he had severe diarrhœa uncontrolled by medication and having no relation to diet. I believe that this symptom, intestinal hypermotility, was another evidence of hyperexcitation of his sympathetic nerves.

To summarize: The goiter, the exophthalmos, the fibrillation with consequent decompensation, and the diarrhœa are the results of disturbances of the sympathetic system. I profess, however, no knowledge as to the fundamental cause of the sympathetic disturbance in this case. Regarding therapy, I am not surprised that digitalis has not had the slightest effect upon the irregular and rapid ventricular activity nor upon the heart failure. We have no specific, nor anything like a specific at present, which can influence continued fibrillation in patients with goiter.[1] The primary therapeutic effort must be directed toward some drug which can control the hyperexcitable sympathetics. I have tried digitalis in several other cases of goiter with fibrillation and heart failure without benefit.

The discussion of Case 92 shows the importance and need of correlating all factors and data in the study of the individual cases of auricular fibrillation which you may be called upon to diagnose and treat. If a case of decompensation is due to auricular fibrillation with mitral stenosis you will find, with few exceptions, that digitalis acts almost as a specific. I prefer to give the tincture as already outlined in a previous Clinic. I want to emphasize again the importance of keeping such cases for an indefinite period of time under the influence of this drug, otherwise they will again decompensate. Case 91 is an excellent example of that fact.

Let us now take an instance of auricular fibrillation in senile cardio-sclerosis with fairly marked irregular and rapid ventricular activity and with signs of decompensation There again digitalis will probably have a beneficial effect. I say "probably" because one must understand that in senile cases there are usually advanced pathological changes in the myocardium, endocardium and the coronaries, thus there is often not enough healthy heart left

[1] The specific action of quinidine was not known at the time this and other cases of fibrillation were presented.

upon which the digitalis can act. Such changes constitute, I believe, the most potent reason for disappointment in the action of digitalis in these individuals. Digitalis may indeed block the discordant impulses coming from auricle to ventricle in fibrillation, and hence help steady the ventricular action; but it cannot well increase the contractile power of a considerably damaged heart.

I shall now briefly discuss some of the salient extra-cardiac causes of fibrillation in patients with otherwise normal hearts. Some years ago I observed a case of fibrillation without dyspnœa in a tobacco smoker with an organically sound heart. A similar case has been reported in the literature. An instance of transient auricular fibrillation has been reported in a laborer who while working in a vat was overcome by hydrogen sulphide gas; his heart was organically normal. We also occasionally observe patients with absolutely normal heart, blood vessels and kidneys, who apparently set up fibrillation as the result of reflex causes arising from stomach or intestinal disturbances. An illustrative case is the following: About two years ago, I was consulted by a lady fifty-nine years of age. She had suffered from belching and regurgitation of food for many years. About ten years ago she had colitis and abdominal cramps. Five years thereafter she had pains over the sigmoid. She was operated upon for appendicitis. Adhesions were found; the pains over the sigmoid persisted. During the past summer she was considerably oppressed by the heat. She went to Atlantic City, and while resting in a rolling chair, she had a fainting spell lasting about five minutes. Her family physician, an excellent and careful practitioner, assured me that the summer before, her pulse had been perfectly regular. I saw her some weeks after the fainting attack; the pulse and heart action were grossly irregular; sometimes slow, sometimes fast. She had attacks of tachycardia during which the heart rate was over 160 per minute; the rhythm was irregular. Only at such times was there slight dyspnœa. At other times the patient complained of a subjective feeling of "palpitation." The gastro-intestinal symptoms consisted of belching, colicky pains and intestinal rumbling. A polygraphic tracing which I took revealed auricular fibrillation. In the absence of any evidence of organic cardiac disease—no decompensation, no edema, no bronchitis, no cyanosis, no hypertension, no abnormal constituents in the urine, no cardiac enlargement, no murmurs, and because of the old though still present gastro-intestinal symptoms, I made the diagnosis of auricular fibrillation arising reflexly from the gastro-intestinal canal. Therapy was directed chiefly to the latter. Bromides were prescribed. A good prognosis was given, although no time limit was set as to the duration of the fibrillation. The patient was allowed to sit out of bed, then to walk and gradually to resume her old activities, even including golfing. The arrhythmia lasted several months. The pulse and heart action finally became regular and remained so. This case is given in detail, partly because of its interest, partly to impress upon you the great importance of obtaining a good, careful previous history, and finally, the

importance of studying, correlating and weighing all data before arriving at a diagnosis.

Returning to Table III, you note that auricular fibrillation may occur as part of or during an attack of paroxysmal auricular tachycardia. The latter means that the auricles are beating regularly at a rate between 180 and 250 per minute. The ventricles may beat at the same speed or not, depending upon whether the ventricle responds to every auricular impulse. When the ventricle does not regularly respond it denotes that heart block is present. It is not unusual to have paroxysmal auricular tachycardia as the result of minor gastric complaints in patients with perfectly normal hearts. Digestive disturbances, in some as yet unknown manner cause abnormal impulses in the rhythm center in the auricle with the resultant production of paroxysmal auricular tachycardia. The occurrence of auricular fibrillation found momentarily in such occasional cases denotes, I believe, that the abnormal impulses have tended to race the auricles at a speed beyond that of auricular tachycardia, so that the auricles can no longer beat rhythmically, and in its place the disorderly activity implied by auricular fibrillation occurs.

Regarding fibrillation from fright and other emotional causes (Table III), I myself have never observed such cases, but they have been reported, and I have no doubt that they occur because the more one deals with graphic methods, the more one encounters irregularities of almost all types. These irregularities in previous years were simply diagnosed as arrhythmias but are now more exactly classified.

Digitalis (Table III) may produce auricular fibrillation. I had one such case under observation several years ago. A boy with a decompensated mitral regurgitant lesion and an otherwise rhythmic heart showed fibrillation every time he was under the maximum effects of the tincture of digitalis. Polygraphic tracings left no doubt as to the type of the arrythmia. I tried the digitalis experiment several times; when the drug was discontinued, fibrillation stopped and the pulse became regular at the end of a day or so; when it was again administered the auricles again fibrillated. It is interesting to note that, though the auricles were fibrillating, the patient felt perfectly well and compensation was completely restored. In this case the toxic action of digitalis upon the vagus was probably responsible for the arrhythmia; at the same time the contractile power of the heart, that is its pumping power—was considerably improved by the drug. It has been shown experimentally that if the entire gastro-intestinal tract of a cat be removed from esophagus to anus, and digitalis be injected intravenously, the cat will go through the action of emesis. In other words, the drug apparently possesses some action upon the cerebral center. Thus, the vomiting caused by digitalis may be similarly explained in patients to whom we have given too much digitalis, or who are particularly susceptible to the drug. It offers a more plausible explanation than the usual one that digitalis causes vomiting by irritating the gastric mucosa.

Referring again to Table III, I now wish to discuss the relation between toxins and fibrillation. Fibrillation occasionally occurs in pneumonia in adult and older individuals, in those with normal hearts, as well as in those with valvular and other cardiac disease. I presume the usual impression of pneumonia toxins is that they possess a destructive influence upon the cardiac muscle, valves and coronaries. In almost every postmortem in pneumonia, there is some cloudy swelling of the cardiac muscle, but it does not differ in degree or kind from that found in other infectious fevers. In my opinion auricular fibrillation occurring during pneumonia is not due to such pathological change, but is caused by some damage to the nervous mechanism —*i.e.*, to the neurogenic control of the heart. In what way toxins may thus act, I cannot tell you. Though we use the term frequently, we possess so little knowledge about toxins and their effects on nerve structures that further discussion along these lines would be purely hypothetical. But it has been my clinical experience (and clinical experience after all is important) that fibrillation occurring in pneumonia in patients with previously normal hearts do not, even in after-years show any damage to the heeart as the result of the arrhythmia. I do not wish to leave you with the impression that pneumonia may not produce wide-spread damage to the myocardium and other cardiac structures. As a matter of fact I believe that this disease is often the *insidious* and first cause of a good deal of cardio-vascular damage which shows itself only in later life. This statement is scarcely susceptible to proof because the process is so insidious and slow. For example in a patient whom I saw some years ago, there was no doubt as to the correlation between a severe pneumonia contracted some years previously and the very gradual onset of cardio-sclerosis. In pneumonia with fibrillation, it seems difficult to conceive of severe cardiac damage which would not show itself immediately after the pneumonia had run its course; in the cases I have observed I could discover no sign of cardiac disease either during fibrillation or immediately thereafter.

In concluding the remarks on fibrillation, I wish again to emphasize the importance of bearing in mind some such scheme as the Tables which give the physiological, clinical and experimental causes. Always try to correlate etiology with the case in hand. In that manner you can attack the therapeutic and prognostic problems more readily and rationally. Such study will also help eliminate mistakes in the application of drugs.

Student: What preparations of digitalis do you prefer and why?

Dr. N.: The action of all the digitalis bodies is alike. I prefer the tincture because it can be given in smaller doses, because of its uniform strength, and because it is fairly stable by reason of its alcohol content. Next I prefer digipuratum or digitan tablets because they are standardized preparations. In an emergency, I prefer strophanthin for its quick effect; one ampule containing one cubic centimeter is injected intravenously or intramuscularly; it should be injected slowly. It is particularly indicated at the onset of severe

decompensation or when auricular fibrillation comes suddenly with signs of circulatory failure, but it should never be administered in cases to whom fairly large amounts of digitalis had already been given. Digalin is variable in strength and hence is not as good as the other preparations just mentioned.

Student: Does the effect of strophanthin last as long as digitalis?

Dr. N.: No, it does not. Its effect lasts 12 to 24 hours; therefore it is necessary to follow it with digitalis. Sometimes it has a most remarkable and beneficial action within an hour or two.

### CASE 93.—AORTITIS WITH ATTACKS OF AURICULAR FIBRILLATION

M. G., male aged 52, is married and has two grown children. He gives the following history: He has never had rheumatism. Twenty-five years ago he had a severe attack of jaundice. Thereafter he suffered from pyrosis and abdominal pain. About five years ago he suddenly was awakened at night by his first "heart attack;" this consisted of a feeling of "thumping in the chest" lasting several minutes. Some years later he was operated upon for appendicitis and purulent cholecystitis. Since these operations he has had no gastric symptoms except when eating fatty food; he then has some belching and pyrosis. Seven years ago he had typhoid fever, and four years thereafter, pneumonia. During the last two years the "heart attacks" are more frequent, occurring even daily. They last much longer than formerly, sometimes for hours. He has been a smoker in moderate amounts but he does not smoke at all now. He is a very poor sleeper; his sleep is often disturbed by erections, by thoughts of business or by introspection regarding his heart. Digitalis in small doses has improved his cardiac condition somewhat. The bromides produce restful nights. He wakes up hungry and feels worse before breakfast when washing and dressing. When not annoyed by "heart attacks," he feels well and is able to attend to business.

**Examination.**—The patient has given us a very intelligent and continuous story of his abdominal and cardiac complaints. The first point to determine is the nature of these "heart attacks." I was fortunate enough to examine the patient during one of the paroxysms. The heart at first beat rhythmically; then, while the patient was sitting quietly, he said, "I am having an attack." The pulse and heart action suddenly became grossly irregular and rapid, with all the clinical characteristics of auricular fibrillation. An electrocardiogram which I immediately took confirmed the type of the arrhythmia. There was no dyspnœa, the patient simply complained of an uncomfortable feeling of "palpitation." The attack lasted several minutes. I was able to decrease the cardiac rate slightly by sudden sharp momentary pressure upon the left and right vagus but the irregularity continued unchecked.

At present the patient, as you see, is perfectly comfortable. He is a very well preserved man for his years. The blood pressure, urine and lungs are normal. The heart and pulse are now regular. Inspection and palpation of the cardiac area reveal nothing abnormal. Upon auscultation, the first

sound over the right base is rough; all the other cardiac sounds are normal. Except for the scars of previous operations, the abdomen presents nothing abnormal. There is slight edema of the legs, as you note by the pitting upon pressure. The orthodiascopic tracing which I here show you presents definite evidence of enlargement of the first portion of the aorta; the remainder of the cardiac outline is normal in size and contour.

**To Summarize.**—The patient, many years ago, suffered from painful digestive symptoms and from "heart attacks." The former were cured by operation. The latter were at first infrequent but during the last two years, they have increased in frequency and duration. Insomnia has been an added symptom, probably because the patient is worried about his attacks.

We have no means of deciding whether the "heart attacks" years ago were of the same nature as the recent ones. From the patient's description of both, the probability is that they are identical. The original paroxysms of auricular fibrillation—for such is the type of the present arrhythmia—may have been the result of reflex excitation of the cardiac nerves from the old dyspepsia. The dyspepsia itself was due to appendicitis and cholecystitis. You gather from the history that even now the patient is sometimes awakened by abnormal hunger sensations.

Why are the paroxysms more frequent of late? The cardiac examination may offer the correct explanation. The rough first aortic sound, the dilated aorta, and the edema of the legs are probably due to aortitis with cardiac insufficiency. The irregular heart action *per se* may also have caused the edema, but the latter seems more likely a part of the composite picture of mild cardiac decompensation. The changes in the aorta are probably comparatively recent, otherwise there would not have been this long remission of cardiac symptoms. The aortitis may have had its origin in the attack of pneumonia three years ago. Such *gradual* progressive changes following pneumonic infections are, I believe, by no means infrequent. They rarely appear immediately after or during the pneumonia, but are one of the late sequelæ. The pathological changes in the aorta—possibly also involving the coronaries—can account for a recrudescence of the old attacks in more severe form. The fact that the patient feels worse upon arising and before breakfast is also of interest. I have observed similar instances in other types of heart disease. It seems due to the fact that it requires an added effort, and is a tax on the weakened cardiac reserve to carry on the circulation properly when the patient first moves about mornings. The hunger experienced in the morning may also cause reflex vasomotor disturbances which deleteriously affect circulatory stability.

Therapeutically I would first reassure the patient. There is no harm in giving him bromides at night for the insomnia. I should also advise him to take a glass of warm milk before retiring so that the stomach be not too empty mornings. He should have a glass of warm milk given to him every morning while still in bed. I should treat him intensively with digitalis for one week,

instead of the irregular dosage he has been taking. For this purpose, he should have two digipuratum or digitan tablets daily for one week. This would have the effect not only of helping the circulation but also of increasing the tone of the vagus. The latter is one of the effects of digitalis. You remember in this connection that I had stated that pressure on the vagus decreased the cardiac rate during a fibrillation attack; the mechanical pressure probably also acted as a stimulus to the vagus.

**Later Report.**—The family physician reported to me that the patient followed the treatment outlined, and that the "heart attacks" have thus far been very infrequent and of short duration.

### CASE 94.—MYOCARDITIS—AURICULAR FIBRILLATION—DECOMPENSATION—THERAPEUTIC RESULTS

M. K., male, aged 45, gives the following history: He considered himself well until two years ago. He is a moderate smoker. There is no history of scarlet fever, rheumatism or of venereal infection. Two years ago he began to cough and to have shortness of breath; later he developed epigastric pains and "palpitation" upon walking. His legs are often considerably swollen.

**Examination.**—You note at once the patient's orthopnœa. Upon inspection of the chest, you observe the diffuse irregular ventricular activity; corresponding to it the pulse is also completely irregular in force and rhythm; in other words the patient has auricular fibrillation. The radial arteries are not thickened. Corresponding to the irregularity in force of the ventricular systoles, the blood pressure of the various beats fluctuates considerably. The majority of the beats registers a systolic blood pressure of less than 150 m.m. There is no pain on precordial palpation. Upon auscultation you hear a soft systolic murmur at the apex; there are no thrills or other abnormal sounds to be heard during systole or in the occasional long diastolic pauses. The liver is moderately enlarged. There is tenderness upon pressure in the epigastrium. There are bubbling mucous rales and sibilant breathing over the chest. At present there is no edema of the legs. The urine contains some albumen with granular and hyaline casts. The orthodiascopic tracing reveals no enlargement of the aorta, but as you see, the left ventricular and right auricular curves are considerably enlarged, giving the cardiac contour a somewhat rounded appearance.

It is evident that the patient is suffering from severe decompensation as shown by the dyspnœa, the auricular fibrillation, and the signs of bronchial, pulmonary and hepatic congestion. The anatomical cardiac lesion is less easy to diagnose. The only abnormal sound is the soft systolic apical murmur. Perhaps, if the heart action can be made more regular and slower by drugs, especially digitalis, we may hear other abnormal sounds not audible now. The renal condition may be the primary disease, although it is more probably the result of circulatory congestion. For the present I believe we must regard the cardiac condition as due to myocarditis, for the following reasons:

The absence of physical signs of an endocardial lesion, the orthodiascopic evidence of ventricular enlargement, the absence of aortic enlargement, and the absence of any rheumatic or other history which would be likely to cause an endocardial lesion.

The results of therapy in this case will practically depend upon what we can accomplish by intensive digitalis and theobromine medication during the next week or two. The patient tells us he has received digitalis, but from his account I believe in insufficient quantity. I shall prescribe the tincture of digitalis in teaspoonful doses three times daily for three days; then he will be placed for two days upon a strict Karrel diet, and take theobromin sodium salicylate in seven grain doses, four times daily. If this intensive medication is followed by good results, the digitalis will be continued in smaller doses, and the theobromin and Karrel days will be prescribed at more frequent intervals.

**Later Examination.**—This patient you saw four weeks ago, markedly decompensated and dyspnoic. The therapy I then suggested had been carried out. As you can see even without a detailed examination there has been marked improvement in the patient's condition. He tells us he feels quite well and comfortable. He is able to walk without dyspnœa or palpitation. The rales in the chest have almost entirely disappeared. The liver is much smaller; there is no epigastric sensitiveness. The ventricular rate is now 60 per minute; it is fairly regular in force and rhythm. Upon auscultation no murmurs are heard. The urine contains no albumen. The average systolic blood pressure is now 160 m.m.

This patient shows the brilliant result of digitalis therapy. He is now in excellent condition and may even do some physical work. He will have to continue taking digitalis, and occasionally theobromin and a Karrel day for an indefinite time, in order to prevent a recurrence of decompensation and dyspnœa. Digitalis in his present condition may be given in ten minim doses, once daily; the theobromin and Karrel diet for two days every month. In the absence of any cardiac murmurs even now with the slow heart action, I believe our original diagnosis of myocarditis is correct.

### CASES 95-97.—TRANSIENT AURICULAR FIBRILLATION

In a previous clinic I demonstrated to you several cases of transient auricular fibrillation. I now have the opportunity of showing you three others.

Case 95. *Transient Auricular Fibrillation Due to Coffee.*—S. K., aged 35, physician, was a heavy smoker in former years but stopped because smoking produced precordial pains. He had influenzal pneumonia some months ago; this was not accompanied by any arrhythmia. The night before I saw the patient, he said that he drank two cups of very strong, almost black coffee, a very rare thing for him. He recalled that the coffee was very strong because it required an exceptional amount of sugar in order to sweeten it. One hour after drinking the coffee, he felt a "fluttering around his heart;" he took his

pulse and found it irregular. He had no dyspnœa and slept well the entire night. He had no subjective symptoms the following day. He kept to his bed for precautionary reasons, simply because his pulse was still irregular.

**Examination.**—You observe that the patient is quite comfortable now, being neither dyspnoic nor suffering from any distress. In fact, to judge from his laughing countenance, he considers the whole matter a joke. The highest systolic blood pressure registered by the strongest beats is 120. This excludes hypertension. I am not going to attempt to find the average blood pressure, a process which requires some calculation because I consider it unnecessary in this case. You note that both pulse and heart action are completely irregular in time and rhythm, the characteristics of auricular fibrillation that I have so frequently pointed out to you. Aside from the arrhythmia, there is not the slightest evidence of cardio-vascular disease; there is no dyspnœa, there are no murmurs, there is no precordial or epigastric sensitiveness, the liver is not enlarged, there is no edema of the legs, the lungs are normal, the urine is normal and there is no history of rheumatic infection.

I believe that the auricular fibrillation is due solely to an overdose of coffee, and not to any organic heart disease. I have never seen or heard of a similar case due to coffee poisoning, but the history is so clear and unmistakable that I think this diagnosis is justifiable. The further progress of this case will probably substantiate or upset the diagnosis. If the arrhythmia be of functional origin and due to an overdose of coffee, the auricular fibrillation should disappear within a day or so, that is, after the coffee has been eliminated from the system. I do not believe any medication is necessary at present.

**Examination Next Day.**—You observe, gentlemen, that the patient's pulse and heart action are regular now and that all the heart sounds are normal. He feels perfectly well. The disappearance of the arrhythmia justifies our original diagnosis, I believe, namely that it was caused by an overdose of coffee.

Coffee is known to cause extrasystoles occasionally, probably by increasing excitability of the cardiac nerves. Although the mechanism of their action may be entirely different, caffein seems also to have an effect upou the heart somewhat similar to that of digitalis, in that both can produce extrasystoles and auricular fibrillation in susceptible individuals.

### CASE 96.—AURICULAR FIBRILLATION FROM FRIGHT

Mrs. D., aged 70, had an attack of influenza some months ago; otherwise she does not recall any recent illness. Yesterday she suddenly became dizzy and fell. This frightened her very much. Soon thereafter her heart began to "palpitate" and there has been "palpitation" ever since. I can get no further details from her.

**Examination.**—You observe, gentlemen, that although the patient looks her years, she has not the thickened radial and temporal arteries common in the old. Nor is she very dyspnœic. Her heart and pulse action are typical

of auricular fibrillation: Both are completely irregular in force and rhythm. The heart sounds, the urine and the lungs are normal. There is no edema of the legs, there is no precordial or epigastric sensitiveness. The heart is apparently not enlarged to percussion.

The question now arises, what is the cause of the sudden onset of auricular fibrillation? Two main probabilities suggest themselves; either this patient may have had a coronary infarct, an accident not so very infrequent in older people and not uncommonly associated with auricular fibrillation; or sudden fright following the dizzy spell and fall may have caused the arrhythmia by some acute circulatory disturbance in the brain, in the neighborhood of the cardio-inhibitory center. I am inclined to the latter hypothesis despite the greater chances of coronary disease in a person of advanced age; indeed, the original attack of giddiness may even have been of cardiac origin. In favor of a functional cause—fright—is the absence of dyspnœa and of gastric disturbances, and the fact that the arrhythmia immediately followed the fall. I shall prescribe digipuratum tablets, one three times daily. We shall re-examine the patient in a day or two; perhaps the further course of the case will indicate the correct diagnosis.

**Examination Two Days Later.**—This patient, you recall, I presented as a case of transient auricular fibrillation. You notice now that the pulse and heart action are normal. There are no murmurs. The patient is quite comfortable. I shall discharge her and let her follow her usual occupation.

I believe that the cause of the auricular fibrillation was fright, for I do not think that digitalis would so quickly have cleared up the arrhythmia, had it been due to a coronary infarct. When the latter interferes sufficiently with the intra-cardiac circulation to produce auricular fibrillation it would almost of necessity cause other cardiac symptoms such as pulmonary edema and precordial pains.

Assuming that the arrhythmia is of functional and not of organic cardio-vascular origin, let us theorize for a moment regarding the possible mechanism which induced the auricular fibrillation. One may, for example, hypothetic-ate that the fall itself, by causing a sudden change of blood pressure, produced sufficient disturbance in the vasomotor tone and in the cardio-inhibitory center to have induced the arrhythmia. Or, subsequent to the fall, there was vasomotor instability, thus upsetting the usual control of this center. Even giddiness *per se* may conceivably reflexly alter the vasomotor tone and thus produce an arrhythmia. I realize of course that we are heaping up theoret-ical assumptions with a meager substratum of facts. But it is a rather common clinical experience that sudden fright, for example, produces severe tachycardia and extrasystoles, and I see no reason why it may not also cause auricular fibrillation in exceptional instances. The more I observe and study arrhythmias, the more I am impressed by the fact that the most bizarre as well as the most common types can follow functional derangements. I have even observed a case of heart block of functional origin. I was able to follow

that patient to autopsy, for he was killed in an elevator accident. Careful macroscopic examination of the heart showed no lesion in the auriculo-ventricular conduction system nor in any other part of the heart.

## CASE 97.—TRANSIENT AURICULAR FIBRILLATION—PERMANENT HYPERTENSION

A. K., married, aged 58, had been a moderate smoker. He had gonorrhœa many years ago. He has not had any recent acute diseases. He had always been athletic. Except for cardiac "attacks," of which this is the fourth, he had never had any cardiac symptoms. The first attack occurred four years ago. The patient believes it was the result of too much swimming. The second he believed followed too much smoking.

For several weeks past he has been worrying over financial matters. One night he suddenly noticed that his heart beat very irregularly, at which time he also became dyspnoic. Prior to this, even though working hard, he never was short of breath.

**Examination.**—This is the fourth day, the patient tells us, that his pulse and heart action have been irregular. You observe that he is not dyspnœic now, although he is lying rather flat. Both heart and pulse are irregular in time and rhythm, typical of auricular fibrillation. When you listen to the heart, you will find that the second sound over the right base is somewhat accentuated; the other cardiac sounds are normal. There is no pain on precordial pressure nor does the patient suffer from any subjective precordial discomfort. The systolic blood pressure of most of the effective beats ranges between 160 and 170. The liver is not palpable. The urine is normal. The Wassermann examination of the blood is negative. There is no edema of the legs. The eye grounds, as reported by the ophthalmologist, show no evidence of arterio-sclerosis. The orthodiascopic tracing being passed around shows moderate enlargement of the first portion and arch of the aorta. The remainder of the cardiac outline is normal in size and form; in other words, there is no evidence of ventricular hypertrophy.

To summarize: This patient has auricular fibrillation now; the other past "attacks" may also have been examples of the same arrhythmia. The definite cardio-vascular abnormalities that the patient presents are an accentuated second aortic sound, an enlarged aorta as demonstrated by the X-ray, and moderate hypertension. I believe that the aortitis is of the slowly progressive type, for there would otherwise not be such long remissions of cardiac symptoms. You see the patient looks very robust for a man of his years and even now, despite fibrillation, he does not feel ill. You may recall that I have already shown you one or two examples of aged individuals with very much enlarged aortas who were suffering from very few cardiac symptoms. Our patient seems to belong to that group. I believe that financial

worry has much to do with the onset of this last pulse irregularity. As I picture the case it seems to me that the neurotic element may have been a factor in causing arrhythmia, and even the hypertension. I shall place the patient upon digipuratum or digitan for several days; for three days, I shall give him two tablets daily, and then for six days, one daily. He is also to receive fifteen grains of the triple bromides every night. I believe the arrhythmia will disappear soon and that the blood pressure will become normal. I shall report to you the further progress of the case at some future time.

**Report Four Months Later.**—The patient took the medication I had authorized. I then sent him to the sea-shore for several weeks. I have seen him often during the intervening four months. One week after the examination, the pulse rhythm became normal and has remained so. The blood pressure varies between 190 and 200 systolic, and from 130 to 100, diastolic. Our examination of the phenolsulphonephthalein output for two hours was 40 per cent. This is the lower normal limit. The patient is again active, but is not rushing about quite as much as formerly. His only complaint is slight dyspnœa upon walking stairs, and slight palpitation. These symptoms seem to have no correlation with the height of the systolic or diastolic pressures. From all present indications the hypertension seems to be permanent. Whether it antedated the time we first saw him, we have no means of ascertaining, because the patient had not had his blood pressure taken for many years.

I scarcely believe that a latent interstitial nephritis plays an important role in the hypertension of this patient because none of the usual earmarks of nephritis are present. Neither the dyspnœa, pallor, anæmia, gastric nor precordial symptoms, eye-ground changes, nor low phthalein output is present. On the other hand I do not wish to diagnose the case as one of so-called functional hyperpiesis, for the enlarged aorta is distinct evidence of some organic arterial change.

**Later Note.**—Some months later, I saw the patient in an attack of moderate dyspnœa with severe precordial pain radiating to the left arm. The temperature was normal. Heart action was rhythmical. The only physical sign in addition to the old murmurs was a localized area of dry pericarditis in the fourth left interspace. This was undoubtedly due to an inflamed pericardium covering an infarcted myocardial area from occlusion of a small coronary. In view of this definite evidence of coronary disease, I believe that the previous attacks of temporary auricular fibrillation may also have been results of coronary stoppage from embolism or infarct. The precordial attacks also clear up, I believe, the cause of the hypertension, for the latter is by no means an infrequent accompaniment of aortitis without clinical evidence of nephritis. Indeed, cases of aortitis with cardio-sclerosis have been described in which postmortem examination of the kidneys both macro- and microscopically did not show sufficient changes to warrant the assumption that the slight nephritis was the cause of the hypertension.

## CASES 98–104.—THE USE AND ADMINISTRATION OF QUINIDINE SULPHATE IN AURICULAR FIBRILLATION

Gentlemen: I have waited several weeks until I was able finally to gather together several cases of auricular fibrillation so that we could observe together the effect of quinidine on the rhythm. Where it was found necessary, the cases have first been properly digitalized, for it is very important to put the patients in as good a state of compensation as possible before quinidine is given. Hence, digitalis is first administered to those who present evidence of heart failure. It will perhaps simplify our problem if I take up briefly our present knowledge of quinidine—its action, dangers, disadvantages, etc.

Since quinidine first came into fairly general use in 1921 because of its extraordinary power to restore the normal rhythm in a certain proportion of cases of auricular fibrillation, there have been case and experimental reports dealing with the fundamental method of action of quinidine. In an excellent paper dealing with the experimental phase, Lewis and his co-workers draw the following conclusions regarding the effects of quinidine sulphate in auricular fibrillation as found in man. They state that the drug has two actions: (1) Direct action upon the auricular musculature by which it lowers the sino-auricular rate; depresses and prolongs conduction time from auricle to ventricle; and lengthens the refractory period of the auricular musculature. The last is of most importance. (2) It causes partial paralysis or release of the vagus. In this it is opposite in effect to digitalis which increases vagal control and inhibition.

I call your attention now to the new fact that auricular fibrillation is regarded as basically due to irregular blocking of a continuous "circus" wave of excitation, the normal method of spread of impulse through the auricles. Although the circus wave still continues, blocking of it depends upon differences and defects in conductivity and irritibility of various portions of the auricular musculature. The consequence is what we clinically know as auricular fibrillation. Before normal rhythm is restored by quinidine, there is usually a significant decrease in the number and irregularity of the fibrillatory waves as determined by electrocardiograms taken by direct chest leads; there is also preliminary stage of increased ventricular rate, whether the normal rhythm be restored or not. This action seems due to release of vagus control, a factor just mentioned. Occasionally the ventricular action becomes very rapid, irregular and violent, in consequence of which there may be marked dyspnœa. This, and the fact that quinidine can cause gastro-intestinal disturbances, such as nausea, vomiting and diarrhœa, and that it has occasionally produced cerebral embolism from sudden slowing of the blood current in the auricles coincident with the restoration of normal rhythm, are possible dangers following the employment of the drug. As an instance of tachycardia, there is a recent report of a case of auricular fibrillation in a

patient with mitral regurgitation who was given quinidine sulphate, 8 grains daily for three successive days. In all 24 grains were given. On the second and especially on the third day, there was evidence of severe decompensation with auricular fibrillation still present. It took thorough digitalization to slowly restore compensation. I wish again to emphasize that it shows that there probably exists a drug idiosyncracy to quinidine in some individuals, as indeed is true of all powerful drugs. Quinidine undoubtedly increases the tendency to decompensation or actually causes heart failure by its occasional action in causing marked increased rapidity of the heart action.

I have had occasion to use quinidine sulphate in hospital practice in patients with auricular fibrillation in all types of cardio-vascular disease. Many were old individuals with marked cardio-vascular and cardio-renal changes, some with, others without hypertension. Many were decompensated. The net result of quinidine administration was temporary restoration of normal rhythm in a few of these cases. A few complained of nausea. One or two vomited. Some exhibited an increase of tachycardia, but none to any alarming extent. Some of the patients had two or three courses of quinidine—but the results were the same. All of the patients were properly digitalized before the quinidine was given. I had not expected permanent restoration of the rhythm in these advanced cases of cardio-sclerotic disease, for it was my clinical impression that quinidine was of value chiefly in younger individuals without far advanced degeneration of the heart muscle, in other words, mainly those with valvular disease. My routine administration consisted in giving the quinidine sulphate in 5 grain doses every hour for three doses. I examine the patient after the first dose to see whether there is any hypersensibility. I again observe the patient after the third dose. If then there are no untoward symptoms—no vomiting, diarrhœa or undue tachycardia—5 grain doses at hourly intervals are repeated for three more doses. Thus altogether 30 grains are given in one day.

I have found this method and dosage satisfactory and have up to the present not noticed any ill effects from this method, although I have often observed some alarming symptoms from the quinidine as such. Two cases I shall later report to you. The method itself seems safe because any important drug idiocyncracy will probably show itself after the first five or after the 15 grains, a dose which seems well within safe limits.

**Case 98.**—Mrs. F., a widow 46 years old, has to support herself and does much laborious work carrying bundles and climbing stairs. She has complained of frequent attacks of bronchitis and dyspnœa for several years. She reached her climacteric two years ago; it was accompanied by sweats, flushes, menorrhagia and rapid heart action. Of late months, dyspnœa has become more marked. She has had "bluish" hands for years. She sought hospital admission chiefly because of her dyspnœa and fainting spells.

**Examination.**—You note that when at rest in bed there is only slight dyspnœa. Her hands and lips are moderately cyanotic. There is no evi-

dence of bronchitis or of pulmonary congestion. She had already received one and one half ounces of the tincture of digitalis. Despite that, you notice that the heart action is still quite irregular both in force and rhythm, the usual clinical evidence of auricular fibrillation. The urine is normal. Most of the beats register a blood pressure around 180, therefore I believe we are safe in saying that hypertension is present. In the fluoroscope I found that the heart was practically normal in size for one of her physique. You hear no heart murmurs on auscultation. There is no edema of the legs. My impression is that the laborious work and nerve disturbances incidental to the menopause are more responsible for her cardic condition than advanced cardio-vascular disease. This is based upon the absence of such physical evidence as is usually found in cardio-sclerosis. The heart is not enlarged; there are no murmurs; there are no precordial tenderness and pains, so significant of serious coronary disease; there is no pulmonary edema or edema of the legs. I have already pointed out to you in other Clinics that women at the menopause are particularily susceptible to arrhythmias and hypertension, especially if there be some pathological changes in the cardio-renal vascular system. These changes, however, need not necessarily be marked or advanced. It is only the correlation and careful observation of all the data—the history, the duration of symptoms, the physical examination—which helps in the determination of how much the symptoms are due to organic changes, and how much to superimposed functional changes. In our present case the fainting attacks may have been due to severe paroxysms of tachycardia from her hard work.

**Quinidine Administration.**—A five grain quinidine capsule had been given about one half hour ago. The patient does not complain about nausea. The cardiac condition is the same as when we saw her before the drug was given. We shall therefore give her two more 5 grain doses one hour apart. . . . Examination after 15 grains of quinidine sulphate. The patient, as you observe, has a heart rate slightly more rapid than previously. I believe it is perfectly safe to go ahead with the other 15 grains this afternoon, to be given in the usual 5 grain doses at hourly intervals. We shall see her again tomorrow.

**Examination Next Day.**—The house physician reports that the patient complained of a little "throbbing" in the head, that she felt nauseated yesterday afternoon and that she vomited during the night. He found the rhythm normal this morning. Let us examine the patient. She says that she now feels quite well. The heart and pulse are perfectly regular, the rate is 75 per minute. The blood pressure is quite different from the previous readings, for the systolic is 100 and the diastolic 80. In other words, at present, she has hypotension, not hypertension. Although the reason for this change is not quite clear to me, it serves to emphasize what I said about the influence of purely functional derangements: Perhaps the hypertension as well as the fibrillation were chiefly functional in origin, hence the drop in systolic blood

pressure when fibrillation ceased and normal rhythm was restored by quinidine. That the low systolic blood pressure is not due to myocardial failure is shown by the fact that the patient is comfortable and has no dyspnœa.

**Later Report.**—The next day the rhythm was still normal, there were however, occasional extrasystoles. I have no opportunity here to take electrocardiograms. It is known, however, that extrasystoles are not uncommon after the normal rhythm has been restored by quinidine. By graphic tracings these have been found to be usually auricular premature contractions. This is another clinical evidence of the after-effect of quinidine on the auricular, rather than on the ventricular musculature. I saw the patient again after two days. She was again fibrillating, the ventricular rhythm resembled what we found at our first examination. This then is an instance in which the drug had but temporary effect. Perhaps patients of this kind should be kept under the effect of quinidine by continuing the drug in smaller doses.[1]

**Case 99.**—Mr. B., aged 54, came under my observation for the first time about one year ago. He was then extremely decompensated and had been so for several months. He presented the usual evidences of advanced heart failure: Cyanosis, pulmonary and leg edema, congested liver, dyspnœa. He had auricular fibrillation. The cardiac lesion was an old mitral regurgitation. He improved remarkably under rest and digitalis, so much so that he was finally able to resume his work as tailor. I had written him to return to the hospital to study with you the effect of quinidine in his case.

**Examination.**—You observe that the patient is not dyspnœic. The cardiac action is irregular, the rate 80 per minute. Most of the systoles come through at the wrist as pulse beats, that is, there is very little pulse deficit. The liver is scarcely palpable; there is no pulmonary edema or edema of the legs. There is a loud systolic murmur over the mitral area. The patient is still taking his digitalis.

**Quinidine Administration.**—We shall now proceed with quinidine sulphate as in the previous case. He had his first 5 grain capsule one half hour ago. His condition is the same as when I examined him early this afternoon. We can safely give him his other two doses. . . . Now after 15 grains there is as yet no change in the condition: No undue tachycardia, no dyspnœa, no nausea. He may therefore receive the other 15 grains in 5 grain doses at hourly intervals. We shall examine again tomorrow. . . . The house physician reported no untoward symptoms except slight nausea. He said that he found the rhythm normal on his morning rounds. You observe that the heart action is rapid although regular. The rate is 110 per minute. The patient is also conscious of his rapid heart action, and says that he does

[1] After the patient left the hospital, her family physician gave quinidine under my direction. Quinidine was administered in daily 5 grain, and later in 5 grain weekly doses after the primary 30 grain dose. In this manner the rhythm has remained normal and the patient is very comfortable.

not feel better or even as well as before the quinidine. This is probably due to the fact that his ventricular rate is more rapid now than when under digitalis alone, for as you know dyspnœa and other symptoms occurring with auricular fibrillation depend upon the resultant rapidity and irregularity of ventricular action and not on the auricular fibrillation as such.

**Later Report.**—The regular rhythm lasted two days, after that interval, auricular fibrillation again set in as before the quinidine.

**Case 100.**—I got the following previous history of this patient, Mrs. J., aged 34, the mother of two children, from her family physician. She has had a mitral stenotic lesion for years. The lesion has always been compensated; the rhythm normal. She is of a high strung temperament. During the last week she has been very busy and active, taking a prominent part at a charity bazaar. She ate very irregularly and was on her feet a good part of the day. The work and responsibility exhausted her very much. Two days ago while at the bazaar in the evening she noticed that her heart became irregular. The physician says it has been irregular since.

**Examination.**—You observe that the patient is somewhat dyspnoic. On examination you will find the typical physical signs of mitral stenosis with auricular fibrillation—heart action irregular in force and rhythm. There is a distinct rough diastolic murmur. The heart does not appear enlarged. There is no edema. Since the patient is not decompensated, I believe we can safely dispense with digitalis and start at once with quinidine.

**Quinidine Administration.**—The patient has been given a 5 grain capsule one half hour ago. You observe that there is no change in the cardiac condition, the irregularity is the same as before. She shall get two other 5 grain quinidine sulphate capsules one hour apart, making in all 15 grains. We shall again examine her then. . . . The rhythm you now observe is perfectly regular, the rate is 70 per minute. The patient has herself remarked the absence of the annoying feeling of "palpitation." Since the normal rhythm has been restored, it will not be necessary to continue the quinidine in this case.

**Later Report.**—I have seen this patient at intervals for two weeks since our last examination. The rhythm has remained regular except for occasional extrasystoles. The patient is otherwise perfectly comfortable. The extrasystoles are regarded as clinical evidence of a tendency for quinidine to lose its effect with a possible return to auricular fibrillation. It may therefore be expedient to attempt to continue the quinidine effect by giving the drug, say 5 grains every second day. If then the extrasystoles disappear, it may be safe to discontinue the drug entirely.

**Case 101.**—Mr. B., aged 29, a shoe clerk, had articular rheumatism seven years ago, since which time he dates his "heart" trouble. He had always been able to follow his occupation without cardiac symptoms till several months ago. At that time he took another position which required some stair climbing. He soon noticed that he suffered from "palpitation"

and shortness of breath. These complaints brought him to the hospital.

**Examination.**—Here again we have a typical mitral stenotic lesion with auricular fibrillation. The sounds over the aorta are normal. The liver is moderately enlarged. There is moderate dyspnœa. He has already been digitalized.

**Quinidine Administration.**—He got his first 5 grain capsule one hour ago. It apparently has not altered the cardiac condition, nor has the patient shown any especial drug hypersusceptibility. . . . Now 15 grains have been given altogether. There is no significant increase in the tachycardia, no nausea or other unusual symptom, hence we shall continue with the other 15 grains at hourly intervals. . . . The rhythm is still irregular. We shall examine him again tomorrow. . . . The house physician reports that the pulse and heart were regular on his morning rounds, and that the patient felt slightly nauseated. You observe that the heart action is now rhythmical, the rate is normal. You also hear the typical mitral stenotic murmur. In addition, it now becomes evident for the first time that there is another valvular lesion—aortic regurgitation—for you can hear the typical diastolic murmur of that lesion not only at the right base but also at the second and third left interspaces. It is therefore evident that the previous irregular ventricular action either masked other murmurs; or what seems more probable that the irregular ventricular action did not allow sufficiently long diastolic intervals for the regurgitant back flow from the aorta to be heard as a murmur. What influence the aortic lesion will have with the now rhythmical heart is problematical. Probably not much, however, for the diastolic pressure is not very low, there is no water hammer pulse, and all the clinical signs point to mitral stenosis as the clinically predominant lesion.

**Later Report.**—The patient has been walking around the ward for two weeks and feels quite comfortable. He says that he occasionally feels his heart "skip." I have been able to corroborate that the "skipping" was due to an occasional extrasystole. This probably means that the quinidine is beginning to lose its effect. I therefore advised one 5 grain quinidine capsule every morning for three days and then a capsule twice weekly. These doses are purely arbitrary, for enough experience has not been accumulated to be able to predict the proper dosage in such individuals. We do know however, that quinidine passes out of the system at the end of two or three days. Hence the occasional necessity for frequent repitition of the drug in order to keep up its effect. I again examined the patient at the end of another week. The heart rate was normal and rhythmical, there were no extrasystoles. He was then discharged from the hospital.

**Case 102.**—Mr. G., aged 41, a painter, was admitted to the hospital several months ago with the same complaint that he has now; dyspnœa, cough, slight edema of the legs. Compensation was restored with digitalis. He has again been digitalized now.

**Examination.**—You note the typical irregularity of auricular fibrillation as well as the typical mitral stenotic lesion. The cardiac rate is 70 per minute, the beats are fairly regular in force. There is no edema of the legs. There are no rales or pulmonary congestion. In other words, digitalis had again restored compensation.

**Quinidine Administration.**—One hour ago he got his first 5 grain dose of quinidine. Since his condition is unchanged we shall proceed with the other 10 grains. . . . The patient complains of some dyspnœa; you will observe that the heart rate is somewhat more rapid and irregular, this probably causes the dyspnœa. I believe that after several hours we may give the other 15 grains. . . . The house physician reported that the patient vomited during the night. The patient complains that he still feels somewhat nauseated. You observe on examination that fibrillation is still present and that the cardiac rate is what it was at our first examination, about 70 per minute. I shall again try quinidine in a few days.

**Later Report.**—A week after our last quinidine attempt, I again tried the drug in our usual method of administration, 30 grains in divided doses of 5 grains each. The effect was the same, at first, increase of tachycardia with some dyspnœa; later, vomiting. The rhythm was not restored to normal. It is safe to again try quinidine in such cases after a week or two because it has been shown that the drug passes out of the system within three days. In some cases I have made repeated attempts, but in a general way it may be stated that if normal rhythm is not restored at the first trial, it will probably not succeed in the other trials.

**Case 103.**—This patient, Mr. F., aged 64, scarcely looks like a promising case for beneficial results from quinidine. His present cardiac condition is very much better than when he entered the hospital, several weeks ago. From his old cardiac history, we learn that for several years he has suffered from recurring attacks of articular rheumatism, for which he was admitted several times to another hospital. Despite these frequent rheumatic attacks he never had any cardiac symptoms until three years ago when his son was drafted for war service. This upset him very much. Immediately thereafter his cardiac complaints began; palpitation and shortness of breath. These finally eventuated into severe and continued heart failure; cough, edema of the legs, constant dyspnœa, palpitation. On hospital admission, the patient was extremely cyanotic and orthopnoic. There were moist crackling rales and sibilant breathing over the entire chest. There was a soft systolic murmur at the right base followed by a diastolic murmur; the latter was not transmitted. Over the mitral area there was a blowing systolic murmur. The blood Wassermann reaction was negative. Auricular fibrillation was present. It took several weeks of digitalization, theobromine sodium salicylate with restricted fluid intake, and various expectorant mixtures to bring about what might be called a fair state of compensation.

**Examination.**—You observe that the patient is still cyanotic and moderately dyspnoic. You hear the aortic and systolic murmur. The liver is still considerably enlarged as the result of venous stasis. There are soft mucous rales to be heard over the lungs at various areas. X-ray examination of the chest shows that the aorta and left ventricle are considerably enlarged. There is no edema of the legs at present. The rhythm is characteristic of auricular fibrillation. The entire cardiac condition may be summed up as rheumatic mitral and aortic regurgitation, myocarditis, emphysema, congested liver and chronic decompensation. Shock and anxiety over his son seemed to be the factors which initiated the cardiac symptoms.

**Quinidine Administration.**—Five grains had been given one hour previously. There are no untoward results, the patient is still fibrillating. We shall give him 10 more grains in two doses and then examine him. . . . Now, 24 hours after the first dose you observe that the heart is perfectly regular and that the patient feels comfortable. The murmurs are the same as before, rales are still present but the patient feels less palpitation. This was a rather unexpectedly favorable result in a patient with such advanced disease.

**Later Report.**—The rhythm remained normal for four days and then fibrillation again began. Perhaps at some future time, I shall again administer quinidine for a longer period in order to try to get a lasting effect.

**Case 104.**—Miss E., aged 59, states that she had "kidney trouble" one year ago. She has been complaining of gastric derangements, chiefly belching, distension after meals and pyrosis for a number of years. The cardiac symptoms began a few weeks ago. These were a sense of oppression in the chest and dyspnœa. Dyspnœa, however, is correlated not with exercise but with ingestion of meals, it is more marked after eating and even directly after swallowing fluids. The house physician states that on admission a few days ago, the heart was very irregular and rapid, the rate was around 110. She was immediately digitalized. The ventricular rate fell to 80 and later to 40; it has been between 40 and 50 since then.

**Examination.**—You observe that the patient is not dyspnœic. The lungs are normal. There are no murmurs over the mitral area. The second aortic sound is accentuated. The urine contains a heavy trace of albumin and a few hyaline and granular casts. The systolic blood pressure is 150, the diastolic, 80. The Wassermann blood reaction is negative. There is no edema of the legs. The orthodiascopic examination shows a prominent aortic arch, no aneurism, and a heart moderately enlarged to the left. Having given you these data, let us pay particular attention to the arrhythmia. It is not as easy to diagnose as in our other cases because the heart action is slow (the rate just now is 50 per minute) and because the heart beats fairly regularly. On listening for a while, however, you will hear some beats that are weaker, as well as occasional definite but irregularly spaced breaks in the rhythm. On carefully examining the jugular in the neck you will find that

there are no jugular pulsations that correspond to the normal auricular pulsations, in other words, the *a* wave is absent; instead of the normal *a-c-v*-waves found in rhythmic heart action there are but two: The *c* and *v*. These characteristics serve to characterize the rhythm here present as auricular fibrillation with slow and fairly regular ventricular action. Perhaps the latter was due to the digitalis.

**Quinidine Administration.**—One 5 grain dose has already been given. . . . Now 15 grains have been given. The cardiac condition is the same. The other 15 grains will be given later. We shall see the patient tomorrow. . . . The house physician reported that the rhythm was regular this morning. It is regular now, for you will find on auscultation that the action is perfectly rhythmical and that the rate is 65 per minute.

**Later Report.**—The pulse remained regular one day. Fibrillation then recurred, but the ventricular rate was then 70, not around 50. Quinidine in the usual manner was again administered and the next day the rhythm was again normal, the rate between 55 and 70. Quinidine in 5 grain daily heart doses was then advised, to see whether recurrences of fibrillation could be avoided.

It is interesting to inquire into the possible correlation between the gastric symptoms and the cardio-vascular condition. The patient apparently has nephritis, aortitis and moderate hypertension. The symptoms are recent and always showed definite correlation between food ingestion and dyspnœa. I have already pointed out to you the intimate nerve relationship between the vagus supply to the stomach and heart. It is well exemplified in our case. It is evident in the marked effect that digitalis had in depressing the ventricular rate. It also accounts for the increased dyspnœa, the feeling of substernal oppression after meals and after cold drinks. The latter presumably cause esophagospasm and cardiospasm, which again reflexly excite the cardio-inhibitory center and cause the referred symptom-complex.

**Comment and Summary.**—Let us now summarize the effects of the quinidine on the seven cases we have studied together. Three had mitral stenosis. Using the word "permanent" in the limited sense of several weeks, normal rhythm was permanently restored in two, it had no effect on the third. There were two cases of mitral regurgitation, one of whom was severely decompensated. There was temporary restoration of the normal rhythm in both. There were two cases of cardio-sclerosis, with temporary restoration of the normal rhythm in both.

With more extended experience, it may be possible later to attain a permanent result in some of these temporarily restored cases. In some forty cardiac cases of all kinds with auricular fibrillation that I have, up to the present, quinidinized, I have only exceptionally seen harmful effects of the drug if the patient is first digitalized and if one has the opportunities to observe the case after the first and after the third dose, in order to avoid unpleasant or possibly dangerous results. Indeed I feel so secure in this

method of administration that I have advised it under the supervision of the attendant physician in those cases of auricular fibrillation that I saw either in office or outside consultation. If the doctor is made aware of the possible dangers and he is told how to recognize them clinically, the drug will gradually assume its proper place not only in hospital practice where it is now almost exclusively used, but also in private practice in the hands of the general practitioner.

There is a possible danger to which I have not as yet alluded. I myself have not had any personal experience with it. An occasional case may be found in which administration of quinidine is followed by a shower of ventricular extrasystoles—ventricular tachycardia. The arrhythmia may not show itself at once; it apparently may come at any time during treatment. Its danger lies in the fact that it may possibly portend and be a forerunner of a fatal arrhythmia—ventricular fibrillation. We have as yet no clinical guide in differentiating in advance those patients who may react in this dangerous and possibly fatal manner.

It may be asked of what advantage is it to the patient with auricular fibrillation who often feels comfortable to have the normal rhythm restored. As a matter of clinical observation most of those with restored rhythm lose the feeling of palpitation due to irregular beating of the heart and are able to do much more work and be more active than when fibrillation is present.

Keeping in mind the exceptional case, and the various dangers and contraindications to quinidine, I believe that quinidine, like digitalis has a large sphere of usefulness in every case of auricular fibrillation. (Contraindications are given in Chapter XX.)

Having summed up the advantages and indicated to you some of the disadvantages of quinidine, it remains to give you the details of the two cases in which quinidine was followed by untoward results.

Mrs. W. age 32, patient of Dr. N. gave a history of frequent tonsillitis. Her tonsils were removed one year ago. Total hysterectomy was performed two years ago since which time she gained 20 pounds in weight. No reason for the operation could be obtained. She had rheumatism one year ago. She had complained of frequent attacks of dyspnœa and tachycardia for some time; they were not always the result of overexertion. The attacks have been worse of late months. On examination the blood pressure was normal. There was a typical mitral stenotic lesion present; the rhythm was disturbed by occasional extrasystoles; electrocardiograms showed them to be auricular in origin. The orthodiascopic tracing revealed moderate enlargement of the pulmonary artery, right auricle and left ventricle. There was no decompensation. Under luminal and extract of suprarenal gland, there was gradual improvement in the dyspnœa and tachycardia so that the patient was capable of doing a moderate amount of walking. The patient was then laid up in a hospital for two weeks with an attack of pneumonia. I saw her again shortly

after she left the hospital. Electrocardiograms then taken showed auricular fibrillation with moderate tachycardia. Twelve digitan tablets were prescribed to be taken once four times daily. These had no decided effect in decreasing the cardiac irregularity. A luminal tablet was then given at night, and for the next day, 8–5 grain quinidine sulphate capsules in two hour intervals was prescribed. Her family physician reported that there was some increase of the tachycardia and dyspnœa. The fibrillation remained uninfluenced. The increased tachycardia disappeared in a day or two. Two days after quinidine was stopped, the patient complained of sudden pain in the right arm and of a cold feeling in the right hand. The radial pulse could not be felt. The blood pressure in the right brachial could not be obtained. It was apparent that she had an embolus in the right brachial artery. The patient passed out of observation.

It is a well known clinical observation that embolism as the mechanical result of breaking off of vegetations of an infected valve, especially in mitral stenosis, is by no means infrequent. It can occur with or without auricular fibrillation. It is not always the result of tachycardia. Some years ago for example I saw a case of embolism of the popliteal artery from mitral stenosis in a patient with no arrhythmia and no rheumatic exacerbation. In our patient, however, since the accident occurred two days after the increased tachycardia from quinidine, it is only fair to assume that the more rapid and perhaps more violent heart action had a marked influence in causing the embolism.

Mrs. K., 74 years old, had been an occasional sufferer from phlebitis for years. Her heart has been irregular for some time; the type of the irregularity as determined by the electrocardiogram was auricular fibrillation. She also suffered from occasional tachycardial attacks at night; these were usually accompanied by gastric distention. There was slight edema of the legs, and mucous rales at both bases. She had been digitalized at various times during the preceding months; this generally controlled the tachycardia and to some extent lessened the edema and pulmonary signs. The orthodiascopic tracing showed an enlarged aorta and moderate left ventricular hypertrophy. The auscultatory signs were those of aortitis, the clinical diagnosis was aortitis and cardio-sclerosis. After she had been thoroughly digitalized for the last time, she was given quinidine sulphate in 5 grain doses according to the method I have already outlined. There were no specially alarming symptoms on the quinidine day. The blood pressure was normal. The next morning the fibrillation had ceased entirely, the pulse and heart action were regular with the exception of occasional extrasystoles. The systolic blood pressure was 130, the diastolic 180. The patient felt perfectly comfortable in every way. Early in the evening the patient vomited and soon thereafter fainted. There were many single as well as grouped extrasystoles; for several minutes at a time there was typical coupled rhythm. After the first fainting attack the patient was taken with generalized convulsive seizures. She became

markedly cyanotic. She was unconscious with widely dilated pupils. There were comparatively long periods of apnœa. The urine contained a rather large quantity of albumin for the first time. There was no special change in the superficial or deep reflexes. The convulsive seizures though less violent than the initial attack, continued at irregular intervals during the night. During the whole of this time the patient was in coma. When I saw her the next morning her mentality was again perfectly normal. There was no dyspnœa. The urine contained albumin and casts. Coupled rhythm was present. My impression was that she had entirely gotton over the serious phenomena of the night before, and that she would probably slowly recuperate. She simply complained of her tongue which she had bitten during her convulsive seizures. The patient died suddenly late in the evening. An autopsy could not be obtained. The cause of the convulsions and circulatory collapse may be attributable to two possible causes: First, the toxic quinidine effect on the cerebrum somewhat similar to what occasionally happens when overdoses of quinine are given. Second, the toxic effect of quinidine upon the heart muscle. In my opinion the latter was the cause of circulatory collapse, as well as of death. It seems to me that the convulsive seizures were probably caused by some secondary cerebro-circulatory disturbance, possibly a temporary thrombosis in the larger cerebral veins. The consequent disturbance of the cerebral blood current may be a conceivable cause of the convulsive seizures, general cyanosis and circulatory collapse. The sudden clearing of the cerebral blood stream may also have accounted for the temporary remission of all the serious symptoms as well as for the normal mentality of the patient on the day following the convulsions. I can scarcely conceive of quinidine acting as a cerebral poison and producing circulatory symptoms, and then suddenly, with the remission of all symptoms for several hours, produce sudden death. The urinary changes (albumin and casts) were probably congestive changes secondary to the acute circulatory collapse.

This case should act as a warning, for, as has been pointed out in a previous chapter (Chapter XX), patients who have venous thromboses of the legs are bad subjects for quinidine and are apt to have disagreeable or alarming symptoms during the administration of the drug; in such patients quinidine should not be given unless there is some very strong and very definite indication such as in our case where digitalis was losing its usual effect in restoring compensation.

### CASE 105.—HEART BLOCK

Gentlemen, this young woman gives the following history. She is thirty-two years old and has had "heart trouble" since her fifteenth year. She has had typical attacks of inflammatory rheumatism and a "weak heart" for many years. She has had three children. Her first two pregnancies proceeded normally. During her third pregnancy, she suffered from rheu-

matism and fever for about two months. She has again had articular rheumatism since the birth of the last child. During the past year her heart feels "weaker" and annoys her more than formerly. She now has "spells" in which she can feel her heart beat very fast and then she becomes dizzy and feels faint. The Wassermann blood test is negative.

**Inspection.**—We see a small, frail-looking woman who is not dyspnoic while resting quietly in bed. Upon inspecting her chest we see quite plainly a very marked, diffuse systolic precordial heave, the lowest and most prominent point of which is actually in the axillary line over the seventh rib. We note further that the ventricular impact occupies about three-quarters of the left chest. We observe in the neck marked vigorous pulsations, some of which are due to the carotids and others to the jugulars.

**Palpation.**—Over the right base there is a very marked rough, readily palpable systolic thrill particularly prominent in the third right interspace about two inches to the right of the mid-sternal line. This systolic thrill is also palpable, but fainter, to the left of the sternum for a similar distance and over a similar area. In the apical region we find a strong diffuse impact with a suggestion of a systolic thrill.

**Auscultation.**—Corresponding to the area where the thrill is most marked there is a loud rasping, rough systolic, and also a fainter diastolic murmur. The former is transmitted to the carotids, and to the aorta in the interclavicular notch. Over the mitral area, besides hearing the boom of the strong apical thrust, there is a thrill-like systolic murmur and a fainter diastolic one. In addition there is occasionally heard in the apical region a short faint sound quite distinct from the other two aforementioned mitral murmurs. These faint sounds come approximately in mid-diastole. The physical signs at the base are characteristic of a double aortic lesion. The murmurs at the mitral area are probably transmitted from the aortic region, athough it is possible that an actual mitral regurgitant lesion is also present. But the prominent physical signs over the aorta together with the tremendously hypertrophied heart indicate unmistakably that the main valvular lesion affects the aortic valves, and that a mitral lesion, if present at all, is of secondary importance.

This patient entered the hospital with a rhythmical pulse rate of 35 to 40 per minute, a rate she has since maintained. You have heard from the patient herself about the "spells" she has had. I want you now to notice that the pulse at the wrist is regular, and that you do not feel any extrasystolic beats at the wrist nor at the apex, as in some cases of abortive extrasystoles which are heard over the heart but missed at the wrist. I also want you to observe that there are two jugular pulsations to every heart beat. You can verify this by placing your finger in the suprasternal notch where you can distinctly feel an aortic thrill. Every time you feel the thrill, you see two jugular pulsations—one comes at about the same time as the aortic pulsations, the other about equidistant between it and a succeeding one. In other words there are two venous pulsations to every arterial one. Since the

jugular pulsations are of course produced by the auricle, and the aortic pulsations by the ventricle, it denotes that there are two auricular to every ventricular contraction. Such an arrhythmia is called heart block with a two-to-one ratio. One can make a diagnosis of heart block readily in this case without graphic methods because the carotid and jugular pulsations are so pronounced and obvious. Probably the above mentioned faint murmur or sound heard in mid-diastole is caused by the auricular pulsation falling between the two ventricular contractions, an auscultatory phenomenon fairly often present in heart block.

And now you observe that the patient's history and symptoms are readily correlated with the physical findings. There is a history of rheumatism, "weak heart," and latterly of "spells." These "spells" are doubtless examples of Stokes-Adams syndrome, common in heart block, and due to cerebral anemia. Probably the attacks of dizziness occurring without absolute loss of consciousness represent mild Stokes-Adams complexes. The patient has not had attacks of any kind while in the Hospital.

Let us now try to discover why this patient developed heart block. The rheumatism unquestionably produced the valvular lesions. Her cardiac condition antedates the last pregnancy by many years. This is evident not only from the history but also from the tremendous cardiac damage. As a result of the last attack of rheumatism, the added pathological "insult" presumably consisted of myocardial inflammation and infiltration (so-called submiliary myocardial nodules or "Aschoff bodies"), in or near one of the component parts of the auriculo-ventricular conduction system. Heart block can come from other causes, for example, from infarcts, gummata, tumors and calcareous infiltration which destroy the junctional tissues, but in this case rheumatic myocardial changes seem the most probable.

The liver of the patient is enlarged; there is no edema of the legs.

**To Summarize.**—This patient has an old double aortic lesion, an old hypertrophied heart, recurring rheumatic attacks, and a two-to-one heart block, the latter probably recent. Incidentally you will have observed that by eliciting a good history, by careful inspection of the chest and neck, and by palpation over the right base and in the jugulum, we have obtained almost complete information of the cardiac status. Auscultation added, so to speak, corroborative and confirmatory testimony.

**Therapy.**—If the cardiac lesion were syphilitic in origin, the heart block might be improved by anti-luetic treatment. In the case before us, however, the old rheumatic history, the very advanced valvular changes and ventricular hypertrophy, and the advent of heart block usually denote the beginning of permanent decompensation. This patient may continue for some time in fair comfort, but she will probably never be able to resume her usual duties and occupation.

With reference to the use of digitalis, if she were dyspnoic or edematous, or if signs of decompensation were now present, I would use the drug and

expect some benefit from it. But actual heart failure is not now present, hence digitalis would probably be of no value. One may try to overcome the heart block by administering large doses of atropine, thus perhaps influencing any functional disturbances arising from the vagus. I observe from the history chart that atropine was used without any effect upon the heart block. I think it advisable to use large doses of salycilate of soda because of the rheumatic history. I would push it to its full physiological limit.

It is still a moot question whether digitalis should be used in incomplete heart block because it is said that the use of digitalis may change an incomplete into the complete form. To obviate this possibility, I give atropine with digitalis; if digitalis is given after meals, give the atropine before meals. My opinion is that digitalis is indicated in all types of heart block when circulatory failure is present.

Student: If heart block is due to infiltration of the bundle of His, how may atropine help?

Dr. N.: Atropine will not help if the only cause of the block is the assumed infiltration of the bundle of His, but some cases of heart block are partly functional and partly organic. Even in heart block of purely functional origin, atropine may have no effect upon the arrhythmia. I had one such case under observation some years ago. Autopsy showed that the heart and the conduction system were normal, yet atropine given frequently in large doses during life had no effect upon the block.

Student: How did you recognize that the auricular was twice the ventricular rate?

Dr. N.: I recognized that from the pulsations in the neck. In the notch you may feel the aortic pulse; at the same time you can plainly see two jugular pulsations to the one carotid. In other words, there are two auricular contractions to one ventricular.

Student: How do you explain the pulsation of the veins in the neck?

Dr. N.: The auricle is in a sense continuous with the jugulars through the intervention of the superior vena cava; when the auricle contracts, there is a reflux flow in the jugulars which becomes visible as a pulsation. Very often in less obvious cases, it is necessary to resort to graphic methods—the polygraph or the electrocardiograph—in order to study and decipher the jugular or auricular pulsations.

Student: In a case of heart block, can you expect the greater enlargement to the right or to the left?

Dr. N.: That depends entirely upon the underlying cardiac disease and not upon the heart block as such. Here the heart block is an added insult to an already tremendously hypertrophied heart, but you need not necessarily get marked hypertrophy in heart block.

Student: What is the mechanism of Ducroziez's sign?

Dr. N.: That depends upon the amount of aortic regurgitation. If with the quick systolic ventricular contractions, there be a marked back-

flow into the ventricle because of aortic leakage (that is, because of aortic regurgitation), the arterial pulse wave is but a momentary one and the arterial walls quickly collapse, a fact clinically recognized as the pistol shot pulse. When heard and felt at the femoral artery, it has received the name of Ducroziez's sign. One may also occasionally feel the hyperpulsation and collapse in the digital arteries. This sign can be best elicited by gently clasping the patient's fingers between your thumb and other fingers.

Student: Is the capillary pulsation due to hypertrophy of the heart?

Dr. N.: No. It is due to the quick filling and collapse of the entire arterial system, even including the capillaries.

In complete heart block the ventricular rate is usually between 25 and 30 per minute, and the auricular rate is between 60 and 80. There are, however, cases of complete and incomplete heart-block in whom the ventricular rate is between 15 and 20, and the auricular rate between 80 and 90.

Student: What would you consider, in a general way, the contraindications to the use of digitalis?

Dr. N.: I do not know of any. If there is circulatory failure which comes from cardio-vascular disease the drug should be used. I am not afraid of hypertension, or of slow pulse rates, or of digitalis poisoning as possible contra-indications. Indeed in hypertension digitalis often reduces the blood pressure, especially when cyanosis is present. Of course, I do not mean that digitalis is always followed by beneficial results.

As another instance of aortic disease, I may mention the case of a man of thirty-eight who consulted me recently. He was in the habit of taking vigorous exercise. He was refused life insurance. He thought he had a slight valvular lesion. As a matter of fact he had a typical Flint murmur, that is, a short pre-systolic thrill at the apex with marked ventricular hypertrophy, yet he had no cardiac symptoms. In that case the etiological factor could not be determined. It is rare to have a patient at that age feel so well with an aortic regurgitant lesion and advanced hypertrophy. I may add that it is in just such quiescent cases that a streptococcus viridans infection is apt to develop in later years.

### CASE 106.—COMPLETE HEART BLOCK

This man, M. H., is 69 years old. He says that until three years ago he was always able to work very hard. He had never previously complained of dyspnœa or of other cardiac symptoms. Then, quite suddenly, he had a "weak spell" and lost consciousness. Since that time he has had such spells very often, occasionally falling down during them. From the inception of these attacks he has been unable to work because of dyspnœa on exertion, dizziness, and a tendency to fall when he walks up stairs. The patient is too ignorant to give us further details. Let us examine him in the routine manner.

**Inspection.**—You note that the patient when at rest is not very dyspnœic. Upon scrutinizing the neck you distinctly observe two types of pulsations. The venous is exemplified in the pulsations of the enlarged inferior thyroid veins as well as in the dilated jugulars which you see plainly bulge in the supraclavicular hollow to the right. The carotid pulsations are slow; their rhythm is occasionally disturbed, as you observe, by premature contractions.

**Auscultation.**—Let us now auscultate the heart. I wish again to impress upon you the necessity of always keeping your fingers on the pulse during the entire time that you listen to the heart. If you make that a part of your routine, you will more readily diagnose the presence of arrhythmias and recognize their type as well.

The ventricular pulse rate is 45 per minute. Some of the extrasystoles (more properly called premature contractions) which you hear at the apex are felt as definite pulse beats at the wrist, while others are not felt at all. This simply means that the weaker extrasystoles do not open the aortic valves sufficiently to propagate a systemic pulse wave. There are occasionally two premature beats to be heard at the apex. Let us again carefully study the neck pulsations and find out whether we can now properly interpret the arrhythmia. When you gently press the thumb and index finger of one hand over the corresponding carotids, you feel a slight thrill with every ventricular systole. Corresponding to the premature beats that are heard at the apex, you feel the extra carotid pulsation synchronous with them. It is of special interest to watch the dilated inferior thyroid veins in this individual. Take notice that they pulsate oftener than the carotids; as you know, these venous pulsations are synchronous with and are caused by auricular systoles. In other words, the pulsations of the neck, which are so prominent, tell us that the auricles are beating much faster than the ventricles. Occasionally you observe that the carotid and auricular components of the venous wave fall simultaneously, producing a summation wave. Expressed polygraphically, the *c* and *a* wave fall at the same instant to make one large wave.

The electrocardiographic and polygraphic tracings show that there is no ratio between the auricular and ventricular contractions, an evidence of complete heart-block

Having studied the arrhythmia, let us examine and auscultate carefully for the presence of abnormal sounds and murmurs. Over the lower precordium and towards the left axilla there is a systolic murmur. There is also a rough systolic murmur over the right base in the second and third interspaces; it is this which is transmitted to the carotids and which we felt as a thrill in these arteries. The apical murmurs and those at the base are heard better when the patient is sitting down. Auricular beats falling between the ventricular systoles are not heard. Such sounds are heard as faint taps in a fair proportion of cases of heart block. When the patient is in the recumbent position, aside from the arrhythmia, there are no visible abnormalities. Upon

having the patient sit up, one sees a poorly defined, diffuse impulse below and to the left of the nipple. Upon precordial palpation, there are no abnormal thrills. The aortic pulsation in the jugulum is not palpable.

The right border of the heart is about an inch and a quarter to the right of the midsternal line; the apex beat is heard most plainly in the 6th interspace about an inch below and one-half inch to the left of the left nipple. The blood pressure which had been previously taken was 200 systolic and 160 diastolic. The liver is enlarged and reaches about halfway between the lower border of the ribs and the umbilicus. There is marked edema of the legs.

How shall we interpret the auscultatory phenomena—the rough aortic and the mitral murmurs—in terms of the cardiac pathology in this case? What lesion or lesions are present? In view of the hypertension, the decompensation (as shown by the enlarged liver and edema), the presence of heart block, the absence of a rheumatic history, the fact that the cardiac symptoms started late in life, and the age of the patient, we may be sure that he has cardio-sclerosis, a composite of myocarditis, and of sclerosis which affects the endocardium, the aorta and the coronaries. The mitral systolic murmur is the result of sclerotic changes. Because of the abnormally slow heart action, there is insufficient blood supply to the brain, therefore cerebral anemia results, and in its train follows the Adams-Stokes syndrome. The latter may vary from attacks of momentary semi-consciousness to rather long-continued absolute unconsciousness, coma and convulsive seizures. The patients rarely die during a paroxysm.

It is interesting to speculate upon the lack of cardiac symptoms for so many years in this individual, and upon the final pathological "insult" which caused heart block. We must of course assume that the cardio-sclerosis had been present for many years prior to the inception of the cardiac symptoms three years ago; for, unless the pathological process can be attributed and traced back to some definite, severe, and comparatively recent infection, wide-spread changes as are here present take many years to evolve. Such data of recent infection are absent. The man was steadily at work until his "spells" began. I believe that the secret of the absence of symptoms in this and similar cases lies in the fact that the pathological process is often a very slow and insidious one; thus cardio-vascular functions are only very slowly and gradually damaged; thus also the cardiac reserve force, the socalled factor of safety, is sufficiently large to accommodate itself to the damage, until the disease becomes very wide-spread, or until some vital area is involved.

What special damage caused the heart block? The "insult" was apparently a sudden one, for until the "weak spells" began, the patient felt well, despite a considerably damaged heart. My assumption is that there was infarction of the small special branch of the coronary which supplies the atrio-ventricular conduction system, thus suddenly interfering with the

latter's nutrition. Such an accident would at once nullify the specific power of the bundle to carry impulses from auricle to ventricle; in other words, heart block results, and the ventricle follows its own slow, idio-ventricular rhythm. Another possible cause of the block would be advanced interstitial myocarditis in the immediate neighborhood of the bundle; this, by pressure, might also prevent proper functioning of the latter. But it seems unlikely here because such patients usually show the classic signs of severe heart failure long before the onset of complete heart block.

How should this patient be treated? The blood Wassermann was negative. This does not absolutely preclude the presence of syphilis unless several examinations should be negative, and an examination of the spinal fluid should also be negative for syphilis. I did not have the opportunity to make such examinations in this individual. I therefore tried the actual "therapeutic test" of giving the patient the iodides and bichloride of mercury; these had no effect upon the symptoms. The importance of making the diagnosis of syphilis naturally lies in the fact that there is much better chance for improvement, possibly a cure. Nevertheless our therapeutic efforts may be balked because the luetic sclerosis and endocarditis may be so far advanced, and scar-tissue be so prevalent, that even vigorous mixed treatment and salvarsan injections have no effect upon the process.

In every case of heart block, atropine injections in large physiological doses should be tried for its effect upon the block. In purely functional cases, or in those without far-advanced disease it occasionally releases the block and produces normal rhythm. I have tried atropine in this patient with no effect.

For decompensation, patients with complete heart block should be treated similarly to those with cardio-sclerosis and normal rhythm—*i.e.*, they should be given digitalis in proper doses. When edema is marked, administration of theobromin sodium salicylate in one half gramme doses, four times daily and restricted fluid intake for several days are also of value. Atropine should also be used in the attempt to prevent the Stokes-Adams attacks. Once these attacks appear, we possess no drug to cut them short, especially when digitalis has already been given.

The prognosis here is very poor. The great chances are that the patient will die within a year or so. Digitalis can help the circulation for a time, but it cannot replace damaged tissue, nor can it have much action on a heart so diseased that there is no healthy tissue left to act upon.

### CASE 107.—COMPLETE HEART BLOCK

A. G., male, is now 69 years old. I saw him for the first time in 1913, with the following history: He had small-pox when a child and gonorrhea 35 years ago. In 1903, following an injury, he had a slow-healing ulcer of the leg. When a young man, he had "indigestion" lasting some years. He had

always been able to work until 1913; he then complained of feeling tired. Actual symptoms first occurred in September, 1912, when he began to have "flashes in the head" while walking, and also complained of general "weakness." In May, 1913, while walking, he suddenly fell unconscious in the street and had to be taken home. He noticed no weakness of the limbs after recovery from this attack. During the same month he had two other weak and dizzy spells, but no unconsciousness. At the time of the first spell his doctor told him that his pulse was very slow; the patient himself had no knowledge that the pulse had ever been slow before this. He has never had any convulsions or twitchings. Abdominal complaints began after his attack in May, 1913. These consisted at first of pains over the gall bladder region and in the epigastrium. He was constipated, never vomited, but was occasionally nauseated. Gastralgia began within one half hour after meals and lasted about two hours; "flashes in the head" usually accompanied these pains. His tongue was frequently coated. After rest in bed for two weeks the gastric symptoms and constipation disappeared, the tongue became less coated and the appetite improved. Dyspnœa was slight and an almost negligible symptom.

My notes of the examination in 1913 are as follows: A well-nourished man, pupillary and other reflexes normal; no dyspnœa, no dependent edema. There were at times vigorous carotid pulsations; jugular pulsations were not prominent. Palpation, inspection and percussion of the cardiac area revealed nothing abnormal. Upon auscultation, there was a loud rough systolic murmur over the mitral area. Over the right base, there was also a rough systolic murmur which was transmitted to the carotids. The aortic pulsation in the jugulum was not abnormally prominent. The ventricular action was slow, regular and usually about 40 per minute; faint, distant sounds were occasionally heard at the apex in the inter-systolic periods. The lungs were normal. The systolic blood pressure taken at different times varied from 180 to 130 m.m., the diastolic from 90 to 50. The urine occasionally contained a trace of sugar; otherwise it was normal. The stools were normal. The Wassermann blood test taken several times was negative and the Wassermann reaction of the spinal fluid was also negative, but showed distinct increase of mononuclear cells. The eyegrounds showed retinitis.

I did not see this patient again for five years. The intervening history showed that except for occasional attacks of indigestion he had felt quite well. There had been no cardiac symptoms or flushes for some years. About a week before I last saw him, he said that after drinking two glasses of cold water there were epigastric pains with almost constant hiccough; since then he has had abdominal discomfort and cough.

To summarize: There had been sudden onset of a slow pulse accompanying an attack of unconsciousness; there were rather frequent vasomotor symptoms (flushes); otherwise there were no circulatory complaints, no dyspnœa, no edema. There was auscultatory evidence of changes in the aortic

and mitral valves. There were painful gastric symptoms which have since disappeared.

Let us proceed with the present examination. This man, now 69 years old, is, as you see, well nourished and able to lie quite flat without dyspnœa. The jugular pulsations on the right side of the neck are somewhat more prominent than on the left, but on neither side are they sufficiently distinct to be accurately counted. You see along the inner border of the right sterno-mastoid the pulsatile rise of the tissues synchronous with the carotid pulsations. Now, as I press upon the root of the jugulars on the right side, the pulsations become more prominent, so that one can count their number fairly accurately. You observe that while the excursion of most of the jugular pulsations is small, there are occasional ones which are more marked. Upon inspection of the chest, the cardiac apex is seen to beat an inch below and slightly to the left of the left nipple, the heart is beating regularly, slowly, and by actual count, at a rate of 56 per minute. There is no pain upon precordial pressure. Upon auscultation a rough systolic murmur is heard over the mitral area, occasionally followed after a slight interval by a scarcely audible sound. Over the right base, a rough loud systolic murmur is heard; this is transmitted chiefly to the right infra-clavicular region. In other words the physical signs are about the same as at my first examination five years ago.

It is very important in the auscultation of a patient with arrhythmia, to disregard the latter for the time being, and to concentrate one's attention on the heart sounds only, for the purpose of establishing the presence or absence of abnormal sounds.

Before we go into the interpretation of the faint sound following the ventricular systole, let us discuss the significance of the loud systolic murmur heard practically over the entire anterior surface of the chest. It is loudest over the upper sternum and tails away toward the mid-sternum; the mitral murmur also diminishes toward the mid-sternum, so that at the latter area there is a small space in which no murmur is heard. I believe this patient belongs to the group of cardio-sclerotics in whom the murmur is due to degenerative and sclerotic changes in the mitral valves, and in the aortic valves and walls. The former causes a regurgitant systolic murmur with the area of transmission just described; the latter, a loud rough and sometimes rasping systolic murmur diminishing toward mid-sternum. I may have an opportunity to show you such cases at a later clinic.

Let us now interpret the little extra sound occasionally heard in the inter-systolic intervals at the apex. It sounds almost like the faint tap of a fetal heart. It is due to the auricular systoles which occur in these periods. In other cases of heart block that I have examined, the auricular sounds were more distinct and audible than in this individual.

The margin of the liver is palpable about two inches above the umbilicus in the median line.

The slow and regular pulse and heart action, the frequent jugular pulsations, the auricular sounds occasionally heard at the apex—all these indicate heart block. The Wassermann reaction of the blood and spinal fluid are negative. There is no reason to suspect a neoplasm pressing on or invading the bundle of His. Besides, the existence of heart block for five years, as in this patient, precludes such a diagnosis. How, then, shall we interpret the etiology of this heart block? With cardio-sclerosis, as instanced in the previous case there is always co-existent myocarditis and often marked changes in the main coronaries or their subsidiaries. We know that the bundle of His has its own separate arterial supply. The question to decide is, has the block been brought on by some sudden change in this arterial supply, or has it been the result of myocardial changes affecting the interventricular septum, along which course the main branches of the bundle of His? Although a positive answer is impossible, I am inclined to the former view because of the sudden onset of the block without any prior history of decompensation.

The orthodiascopic tracing of this patient shows some enlargement of the first part and arch of the aorta, and moderate left ventricular hypertrophy. The electrocardiogram shows complete heart block with a ventricular rate of 45 and an auricular rate of 80 per minute. The patient recently had some evidence of decompensation—mucous rales at both bases and edema of the legs. Both have disappeared under the use of digitalis and theobromin sodium salicylate. You see that despite the long continued heart block and his age, the patient looks exceptionally well preserved, and is certainly no worse off than a good many other patients with similar cardio-sclerosis and rhythmic heart action.

Student: What is the etiological factor of the entire condition? Is it specific?

Dr. N.: I do not believe it is specific, because several Wassermann tests, carefully done, were found to be negative. We know, as a result of post-mortem examinations, that there are other changes, mostly of an arterio-sclerotic, non-luetic nature, in or near the auriculo-ventricular conduction system, which may also cause heart block. Besides, the patient had been placed upon vigorous antiluetic treatment, with no effect upon his condition. Atropine sulphate in large doses likewise had no effect upon the arrhythmia.

Student: What has been the primary agent in this case of cardio-sclerosis?

Dr. N.: I do not know. Such changes are so slow and insidious that it is often impossible to ferret out the primary factor. Infections are frequently the starting-point of cardio-scleroses. This individual had small-pox when a child; the pit-marks are still evident. In the absence of other infections and factors, even such an old infection cannot be positively excluded in the etiology. Aside from the arrhythmia, his cardio-sclerosis is no different from that found in a great majority of individuals with this lesion. We are at present very much in the dark as to the cause of the so-called senile changes

in the arteries. We do know that certain conditions are especially prone to produce change in the coronaries and in the cardiac musculature—syphilis, alcoholism, nephritis, are the chief of these; articular rheumatism in the aged is another. Abnormal metabolism with abnormal amounts of non-protein nitrogen in the blood is another possible cause. Infections can bring on changes slowly, which may not be noticeable for years after the original attack has run its course. That is true, for example, of pneumonia. If examined soon after the pneumonia, there are no evidences of any cardio-vascular changes, yet within two or three years there may be recognizable signs of this disease. I have seen several characteristic instances in which I was positive that an old pneumonia was the etiological factor of a marked cardio-sclerosis.

### CASE 108.—HEART BLOCK AND AURICULAR FIBRILLATION, WITH POST-MORTEM SPECIMEN. COMMENT ON THE ETIOLOGY OF AURICULAR FIBRILLATION

Gentlemen: This woman, aged 80, was admitted to the ward Aug. 7, 1920. Except that she entered the hospital acutely ill and that she had been in normal health prior to admission, no history was obtainable which threw any light upon the present illness.

The house physician made the following notes: "Shortly after admission, the pulse rate was around 150 and very irregular. The patient was cyanotic. She was given 1 drachm of the tincture of digitalis and $\frac{1}{100}$ gr. nitroglycerine every five minutes for one hour. The total amount of the tincture thus administered was $1\frac{1}{2}$ ounces. After a short time, the cardiac rate dropped to 40 per minute and gradually became slower until the rate was 28 per minute. It remained at that rate for a few days, to again gradually increase to 40 per minute. Upon admission, she had an attack lasting two or three minutes during which breathing almost entirely ceased, and pulse and heart action were scarcely perceptible. These spells were rather frequent during the first two days; they became less frequent, and disappeared entirely on the fourth day." The patient died Nov. 15, 1920 of a terminal pneumonia.

I examined the patient for the first time about one week after admission. The heart action then was slightly irregular, the rate, 45 per minute. On succeeding days and until the time of death, the ventricular rate remained between 40 and 50 per minute. As a terminal illness the patient developed lobar pneumonia, with fever ranging to 104°F; during this period, the pulse varied from 40 to 65 per minute.

Examination: At my first examination a few days ago, the patient was in the same condition as you see her now. You note that there are no cardiac murmurs. There is no edema of the legs. There are a few mucous rales at both bases. The ventricular rate is 45 per minute and fairly regular. The patient is fairly comfortable and not very dyspnœic. I took the first electro-

cardiogram two weeks after admission (Aug. 21), and then another on Nov. 10, one week before death. Both were alike and showed auricular fibrillation and heart block (Fig. 300).

Because the patient, a city ward, died without leaving relatives or friends, the heart could not be obtained for several weeks after her death. A bit of ventricular musculature was then excised, hardened and sectioned to discover

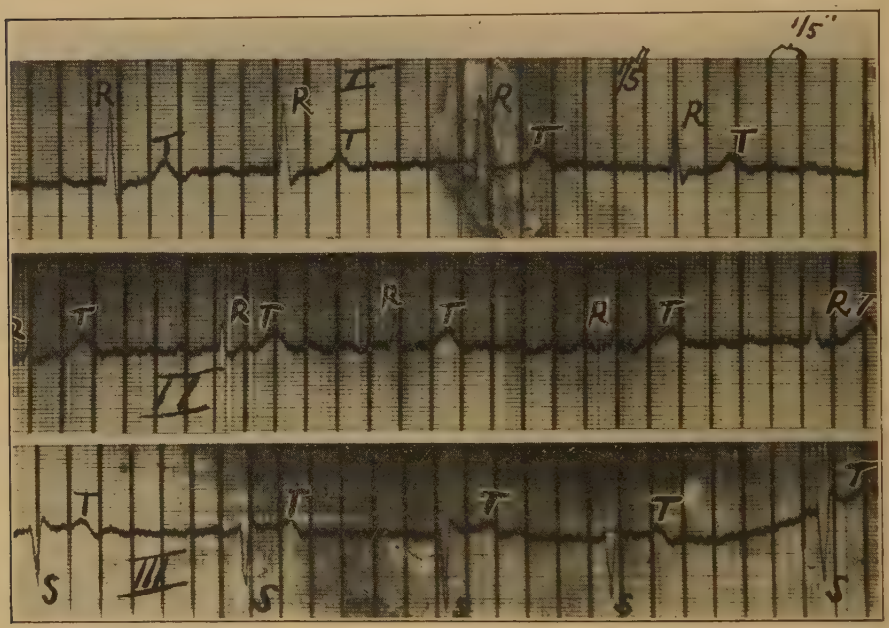


FIG. 300.—Typical section of a long electrocardiogram showing auricular fibrillation and heart block. The ventricular action is slightly arrhythmic, the predominating rate is 46 per minute.

whether the heart could still be used for microscopic study; it was found that the musculature was in too poor a state of preservation for such purpose. You see the heart here. The gross pathology is quite apparent (see Frontispiece) and it is questionable whether microscopic examination would have added anything of importance. The heart is slightly enlarged. The wall of the left ventricle is slightly thicker than normal: The right ventricle is abnormally thin only toward the apex. The right ventricular cavity is somewhat dilated. There are small areas of thickening in the mitral cusps. You note especially that there is surprisingly little interstitial fibrotic change throughout the entire musculature. The auricles are not enlarged or thickened. The region of the sino-auricular node—the pacemaking area at the junction of the superior vena-cava and right auricle—shows no macroscopic change: The artery of that node is not visible or palpable.

The chief pathological changes (see Frontispiece), undoubtedly the ones that finally caused the cardiac disturbances, you observe are found in the coronaries, in the artery of the auriculo-ventricular node (the node of Tawara) and in the musculature surrounding the node. The coronary is thickened and sclerosed, although slightly patulous. The most important pathology lies in the artery supplying the auriculo-ventricular node, the beginning of the auriculo-ventricular conduction system, the normal conveyer of impulses from auricle to ventricle. That artery you note is completely calcified. The artery is seen occupying a position somewhat lower than the normal site of the node, but surrounding the artery (and probably as the result of its calcification) may be seen a distinct zone of degenerated musculature which undoubtedly includes the node, either in whole or in part.

**Comment.**—It is of great pathological and clinical importance, especially in those suffering from what I may term impending "coronary failure," to note how long the final pathological "insult" can hold off before complete occlusion of the coronaries or their subsidiaries occurs. Undoubtedly the calcifying process in the coronary and particularly in the artery of the node has been going on for many years, yet the patient at the time of her death was 80 years old and had lived in cardiac comfort until sudden heart failure supervened; in all probability the latter resulted from final complete closure of the calcified artery supplying that node. This patient is but another illustration of the fact that the *slow* process of intracardiac arterial disease had never at any time sharply interfered with the factor of safety of the heart, and that ample time had probably been given for establishment of coronary anastomosis; for, contrary to older teachings, it is now known that the coronaries are not end arteries and that many deeply seated anastomoses exist.

Before proceeding to the clinical aspect of the case, it may perhaps be well to recall certain pertinent physiological attributes of cardiac musculature. One of these is cardiac irritability. This does not depend upon the strength of the stimulus. It is however considerably influenced by other factors, especially by the state of nutrition of the cardiac musculature at the time of stimulation. Cardiac tissue is refractory to all stimulation during its period of contraction, hence the heart is irritable in the physiological sense during its diastolic or resting period alone. Conduction, another important attribute, varies considerably in different parts of the heart; it is best in the specialized tissues, the sino-auricular node (the rhythm center) and the junctional tissue (the auriculo-ventricular conduction system).

The chief clinical interest in our case centers in the occurrence of heart block and auricular fibrillation, but especially in the etiology of the latter. The heart block was the cause of the slow ventricular activity. This is readily explicable from the gross pathological changes: The calcified nodal artery and the myocardial changes in and near the atrio-ventricular node. These focal changes undoubtedly destroyed the conducting function of the atrio-ventricular node. The massive digitalis therapy—1½ oz. of the tincture

within one hour—may have been a factor in continuing the slow ventricular activity, although there is a definite note of the house physician stating that there were attacks of apnœa with scarcely perceptible heart action (Stokes-Adams syndrome) upon admission. Nor does it seem probable that digitalis, even in such massive doses, could have continued a depressant action upon the ventricles during the remainder of the patient's life, approximately three months.

Regarding the auricular fibrillation in our case, we have no exact data as to its onset, except that the rapid and irregular pulse at the time of admission was probably indicative of this type of arrhythmia. Whether it had been present before hospital admission, we do not know; from the obtainable history, however, there is no evidence of any prior attack of dyspnœa or heart failure. Toxic causes (such as are encountered, for example, in exophthalmic goitre), reflex disturbances, etc.—need no consideration here as possible etiological factors of the auricular fibrillation.

The possible causes of auricular fibrillation with heart block in this case may perhaps best be discussed under the following headings:

1. Structural changes in the pacemaker.

2. Primary nutritional disturbances in the pacemaker.

3. Interference with and blocking of the normal spread of auricular impulses from a normally functionating pacemaker.

1. Structural changes in the pacemaker—Several observers (Cohn and Lewis, Falconer and Dean, Draper, H. Freund and others) had previously made careful serial microscopic examinations of the pacemaker and of the pacemaking area in cases of auricular fibrillation with and without heart block, and had ascribed decided etiological significance to more or less extensive pathological changes in the sino-auricular node. Such changes—usually degenerative and sclerotic in nature—where marked, undoubtedly may be predisposing causes of auricular fibrillation. In some cases of auricular fibrillation however, the sino-auricular nodal region was found normal or almost so. Hence where marked sclerotic changes are absent, other causes for this arrhythmia must be sought. You recall that in our specimen a macroscopical examination showed no evidence of any gross or extensive change in the pacemaker.

2. Primary Nutritional Disturbances in the pacemaker—This would here refer practically to calcification and consequent occlusion of the artery of the pacemaker, or to occlusion of one of the coronaries which occasionally gives off a branch to this node. The former change was absent in our case. Regarding the latter, while the coronaries were thickened and sclerosed, they were still patulous, and hence would scarcely have interfered sufficiently with the arterial supply to the pacemaker, even assuming that a branch of the latter was given off by one of the coronaries.

3. Interference with and the blocking of the normal spread of auricular impulses from a normally functionating pacemaker. Garrey quotes Porter

as stating that "fibrillary contractions may be due to interruption of the contraction waves." Working independently, Garrey and Mines have shown experimentally that if the auricles be thrown into a state of fibrillation by direct faradization (the usual method), and if then a portion of auricular tissue (*e.g.*, the auricular appendix) be temporarily clamped off, fibrillation will continue in the remainder of the auricular tissue. When the clamp is removed, the entire auricle again fibrillates. Garrey and Mines have also found that if the auricle be incised trouser-fashion so that sufficiently broad auricular bridges remain, and then the auricle be faradized anywhere, contraction of the various auricular strips will continue long after faradization has ceased. Garrey calls such contractions "circus contractions," for he assumes that such an excitation wave travels in a continuous circuit, and that the impulse spreads from fiber to fiber. In the experimentally slit auricle, these circus excitation waves are irregularly and sinuously blocked because of the difference in refractory periods of the incised fiber groups and because impulse conduction is interfered with. Thus while the circus excitation as an excitation impulse continues, the auricular strips are actually contracting at varying times and with varying energies. The result is incoordinate contraction, the irregular auricular tremor characteristic of fibrillation. Lewis and his co-workers have recently corroborated Garrey's observations in a series of experimental and clinical papers.

The theory of Garrey and Mines seems to explain some entities in which auricular fibrillation occurs frequently and in which auricular mass as such may act as a factor. Thus, in rheumatic mitral stenosis in which auricular hypertrophy and distension is common, and in cardio-sclerosis in which accompanying sclerotic changes in the auricular musculature are not infrequent, simple mechanical interference with the propagation of a "circus wave" may finally throw the auricle into fibrillation. My tentative interpretation in such instances is that there occurs mechanical hindrance to the spread of the excitation wave similar to what happens in the experimentally slit auricle: The excitation wave cannot spread evenly and ripple-like because auricular conductivity and irritability are interfered with by the diseased or distended auricular strips, hence the excitation wave is irregularly and sinuously blocked and fibrillation results.

In addition to pathological or mechanical changes in the auricular musculature, and excluding all toxic causes, it seems of clinical importance to attempt a correlation of the theory of "circus waves" to disease primarily found not in the auricles but in the ventricles. It is for example no uncommon experience to find auricular fibrillation as a temporary or terminal arrhythmia with or without slow ventricular action in cases of coronary disease during an attack of occlusion (coronary failure) with or without pulmonary edema. Such sudden disturbance in intracardiac circulation with resultant impaired nutrition in the auricular musculature would seem sufficient to interfere with and impair the normal physiological rhythmic excitation wave

in the latter by producing local differences in the refractory periods and conduction time of the auricular excitation wave: Thus a patho-physiological basis for auricular fibrillation would be laid. In our case there was coronary disease with occlusion, the ventricles were beating automatically (heart block) and the auricles were fibrillating. The possible influence of digitalis has already been discussed. Ordinarily in heart block with coronary disease, the auricles beat regularly and approximately at a normal rate. My assumption in such cases is that auricular nutrition is not sufficiently damaged to prevent the normal spread of impulses and excitation waves. Where auricular fibrillation occurs with heart block, the assumption seems fair that auricular disease or at least poor coronary circulation (coronary failure) so damages the entire intracardiac nutrition—auricles as well as ventricles—that auricular excitation waves are irregularly blocked similar to the blocking of the experimental circus waves. As a result the auricles are thrown into that state of fibrillary contraction we designate clinically as auricular fibrillation.

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